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"EGYPTIAN OPHTHALMIA"

BY

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FOR many decades an enthusiastic controversy has been waged over the answers to the twin questions "When and how did trachoma become a disease of considerable extent and importance in Europe?" and "What was the nature of the ophthalmia with which the European armies taking part in the Egyptian campaign of 1798 were stricken?" Anyone who is going deeply into the subject for the first time must be amazed at the mass of relevant literature demanding his attention; and a study of it reveals the fact that opinions differ so widely that, on the one hand, one is told that trachoma "arrived" in a recognisable and even typical form after the Napoleonic campaign, and on the other, that the ophthalmia from which the troops most assuredly suffered was a muco-purulent conjunctivitis of Koch-Weeks' or gonococcal type.

Let us examine a few opinions on this subject, both those of the authors of standard text books, and those of authorities on the particular disease.

"... the affection ... was termed 'Egyptian Ophthalmia' because it was disseminated by soldiers who had acquired it in that country."

TREACHER COLLINS.

" The disease (Trachoma) was not particularly brought to the notice of European surgeons and medical research until practically the entire French Army of 32,000, and to a less extent the British, fell a victim to it during the Napoleonic Campaign in Egypt in 1798-99, and immense numbers of them were sent home ' blind.' From that time dates its introduction to England."

DUKE-ELDER.

" En tout cas, bien que la chose ait été longuement discutée . . . il est plus que probable que les armées de Napoleon et cettes des Anglais, à leur retour d'Egypte, aient contribué à propager la maladie, en particulier dans le nord-est de la France, en Belgique, et en Angleterre."

CUÉNOD ET NATAL.

" Although therefore, trachoma was well known in Europe before Napoleon's time, yet its extraordinary dissemination in the French, English and Italian armies must undoubtedly be attributed in great measure to their infection in Egypt. On the other hand, there is no doubt that the armies of other European nations remained almost entirely free from Egyptian Ophthalmia, in spite of their frequent intercourse with the French troops."

" From the description of the disease given by the authors during the first decade of the last century, there can be no doubt that Egyptian Ophthalmia included not only the trachoma of the present time, but several quite different diseases, such as simple catarrh, follicular swelling, and follicular catarrh, blenorrhoea."

" These incontestable facts refute the view . . . that the transmission of trachoma into every army and country in Europe was entirely the result of infection from the French Army in Egypt. The latter, on the contrary . . . did not suffer to any appreciable extent after its return from Egypt, and within the next ten years it marched through almost the whole of Europe without producing demonstrable epidemics of trachoma anywhere."

BOLDT.

" Never within the memory of man was any army, even the French in Egypt, so terribly ravaged by this disease (trachoma) as the Prussian Army during the years from 1813 to 1820."

EBLE.

" The subsequent history of large numbers of British and European troops showed that after the acute conjunctivitis had subsided the chronic disease, trachoma, made its appearance. When these troops were disbanded they naturally propagated trachoma among their families, and by these means the disease is said to have become more widespread than before."

MACCALLAN.

" The ophthalmias affecting the French, Turkish and British Armies in Egypt during the campaigns from 1798 to 1802 and in 1807 were . . . . the same as those which are still prevalent in Egypt. That is, a combination of Koch-Weeks', gonorrhoeal and post-gonorrhoeal conjunctivitis with trachoma."

MEYERHOF.

". . . . trachoma first obtained particularly *wide prevalence in Europe* in the beginning of the nineteenth century. The French and British armies in Egypt were taken down with it, so that a very large proportion of the soldiers were affected, and when returned to their homes caused a rapid spread of the disease which was hence called ophthalmia militaris. Added to this in the case of the Russian, Prussian and Austrian armies was the enlistment of infected recruits from the eastern countries of Europe, where the disease had always

been prevalent (Boldt). So it happened that when in the Napoleonic wars the armies came repeatedly in contact with each other and with the civil population, the disease became more widely disseminated and occurred in epidemics. In some countries it became frightfully prevalent. In the English army, during the year 1818, there were more than 5,000 on the invalid list who had been rendered blind as a consequence of trachoma. In the Prussian army, from 1813 to 1817, 20,000 to 30,000 men were attacked with it; in the Russian army from 1816 to 1839, 76,811 men were subjects of the disease. In Belgium, in 1840, one out of every five soldiers was affected with trachoma. The French army, which was formerly supposed to form the starting-point of the disease, was just the one that, relatively speaking, was least attacked. The armies disseminated trachoma among the civil population through the discharge of soldiers affected with eye diseases, through the quartering of troops, etc. When they had so many trachomatous soldiers in the Belgian army that they did not know what to do, the Government applied to Jungken, who had then great repute as an ophthalmologist in Berlin. He recommended them to dismiss the trachomatous soldiers to their homes. By this fatal measure trachoma soon became diffused in Belgium to an extent observed in no other European state."

"According to the descriptions of that time, trachoma then ran a very acute course, and was attended with profuse secretion, circumstances which explain the rapidity with which the disease spread and the frequency with which it ended in blindness due to suppuration of the cornea. Now that epidemics have ceased, these acute cases are rare, and the disease has become comparatively benign."

"Trachoma, therefore, not only appears under a varying aspect, sometimes acute and threatening, sometimes chronic and mild, but it also seems to have *changed its character in the course of time.* The explanation of the way in which this has occurred was afforded by the study of trachoma in its native land, Egypt, where ophthalmia Ægyptiaca still rages as in the olden time."

". . . . many of the natives are attacked during the hot season by an acute inflammation of the eyes, in the secretion from which there is found one of the germs causing acute conjunctivitis, and most often the Koch-Weeks' bacillus. It is cases of the former kind that by causing suppuration of the cornea produce the blindness that is so prevalent in Egypt. Now all these different inflammations of the eyes were lumped together under the name of Egyptian ophthalmia and were regarded as identical with trachoma, while as a matter of fact, only the chronic cases are pure trachoma and most of the acute cases are mixed infections. A knowledge of this fact leads us to suppose that in Europe, too, during the great trachoma epidemics, with their cases that ran such an acute and dangerous course, it was generally a question of *mixed infections,* being so either from the outset or from the subsequent superaddition of a second infection (Morax)."

FUCHS.

"If the disease existed at all in England, it seems to have been entirely overlooked till the summer of 1804, when it appeared with alarming violence, in the second battalion of the fifty-second regiment, at that time stationed with the light infantry division in barracks near Hythe in Kent. Not a man of this battalion ever had been in Egypt, and it was entirely composed of a body of volunteers received in one draft from the Irish militia, and very shortly after their arrival at Hythe the disease made its appearance."

"I have reason to believe that a purulent ophthalmia is by no means unfrequent among the peasantry of Ireland . . . . It is a curious coincidence that such a disease should be more peculiar to that country than to this, and that in Ireland also the germ of the Egyptian affection should have been preserved and fostered into action, when little or no appearance of it had taken place in this country. . . . . had I not, by frequent opportunities afforded me of examining the disease imported from Egypt by the second expedition, been able to establish its identity with that consequent on the first, I should have attributed the dreadful prevalence which ophthalmia has had in the Army to the communication of a disease of Irish extraction."

VETCH.

There are probably many factors which combine to account for this extreme diversity of opinion. Perhaps the most important of these is the fact that, in considering the problem, one is tempted to discuss a disease or diseases described over a hundred years ago in the light of experience which has been acquired in the interim, which experience has brought its inevitable increase in knowledge, refinement in diagnosis, and accuracy in terminology. Many contemporary writers applied the term " ophthalmia " to the disease or diseases which they were describing, and many later writers have discussed such disease—whatever its true nature has been—as being identical with one particular clinical entity of the present day. Such an assumption is bound to lead to confusion and even to actual error.

Before considering the question of the actual nature of this post-bellum ophthalmia, it is reasonable to turn one's attention to another possible factor in the production of ophthalmic casualties —that of factitious ophthalmia. This aspect of the question was brought to the notice of the present writer by Mr. H. L. Eason, who sent him a copy of a letter, written by Dr. William Fergusson (not Sir William Fergusson, the surgeon, as Mr. Eason states), then Inspector General of Military Hospitals. The original of this letter was lately presented to the Library of Epsom College by Mr. A. W. Ormond; the present writer is indebted to the Librarian of Epsom College for his courtesy in allowing him to examine this original, of the authenticity of which there is no reasonable doubt.

The letter is of absorbing interest from many points of view, but especially for our present purpose; and since it appeared to be very little known, it seemed a pity to allow it to languish in relative obscurity. It is accordingly reproduced below in full, unaltered except for some minor corrections and additions in the matter of punctuation.

PORTSMOUTH, November 25th, 1809.

Sir,

I have read with great attention the Plan offered by Staff Surgeon Vetch for the prevention and extirpation of Ophthalmia throughout the Army. I acknowledge Dr. Vetch's talents, and feel much respect for the zeal and assiduity which he has manifested during so long a period in the investigation and treatment of this new malady. I confess, however, that all my views of the Disease, founded on perhaps a wider field of experience, but I own of far less minute investigation, differ decidedly from those of

my ingenious friend. I shall endeavour to illustrate these by submitting a short narrative of what I have actually seen, and from thence deduce some inference and offer some observations.

In the year 1806, I first saw this disease in its new form at Plymouth; about 60 Patients from the Armament under Sir Samuel Achmuty having been brought into Hospital with it, where they continued under my inspection for several months. I had then no doubt of the disease being real, tho' I had the disgusting conviction that imposture was also attempted, several men having actually been detected in injuring their eyes by corrosive application, and punished accordingly. The sufferers were mostly young men, nearly without exception recruits from the Irish Militia, and, I need not say, had never been in Egypt; they remained under my inspection about five months. Few of them got well, and several were discharged totally blind, and in that time I had ample opportunities of knowing that the Disease could not possibly be contagious thro' the medium of the atmosphere; I had even reason to believe that it was not infectious from actual Contact or inoculation.

In the year following, I was removed to Kent, the nurse and parent of Ophthalmia. I was still a believer in the real unfeigned nature of the Disease, but could not help there remarking that its victims were for the most part the newly enlisted Irish, that it broke out in an unaccountable manner amongst men marching down to embark, and adhered with particular obstinacy to Regiments such as the 43rd and 52nd, where the system of Drill to the Soldier was severe and incessant.

Early in the following year I embarked with Sir John Moore for Gottenburgh, where the 79th Regiment, on the occasion of Night Watches and Guards being enforced, relapsed into Ophthalmia, and produced some alarming cases; these were speedily cured, not by Medical treatment, but by the determination of Brigdr. General Cameron in directing a system of ridicule and punishment by which their watches and guards were doubled and their indulgencies of grog etc., abridged. He adopted this system with the greater confidence, because that Regiment when at Wheely in Essex, had suffered very severely from Ophthalmia, and had even been deemed incurably affected till a discovery was made of the men having purchased all the corrosive sublimate of the place, under pretence (as they informed the Apothecary) of mixing it with their heel ball.

My next experience in Ophthalmia was of a very different and much more interesting nature. Major General Pagut became my patient, when we returned to Portsmouth, for an affection of one of his eyes which he had contracted in Egypt; it was most painful and intractable, became rather worse during the passage to

Portugal so as to cause serious apprehensions of the eye being lost. When we came to land, however, at Maccira's Bay, he threw aside his shades and washes and encountered in that Climate the heat and dust of a painful march. I considered him as ruined, but to my utter astonishment he was perfectly cured, and I believe has remained well ever since. The Division which he commanded (the 1st Batton. of the 52nd and 2nd Batt: 3rd and 95th) had long been Ophthalmically affected. The hint was not lost; all the patients were set a marching and medicaments were discontinued with an effect precisely similar.

I heard no more of Ophthalmia till about the beginning of December in the same year, when the 2nd Batt; of the 31st Regmt. came to Lisbon from Corunna with about 200 patients, all similarly affected as those I had formerly seen in Kent. The alarm at Lisbon was naturally great; at first they were kept in a state of separation from the rest of the Garrison on board ships; but the disease did not abate, and afterwards they were landed on the opposite shore of the Tagus, apart from all other troops where it even increased. In this Regiment the imposture was more impudent and glaring than I had ever before even suspected. A man for instance would be ordered for guard or for Drill, and while supposed to be preparing his accoutrements would come back with his eyes highly inflamed and thereby be sent to Hospital, where the supposed disease ran its course of inflamed eyelids, ruptured cornea, etc. The Regiment was expected to march up the Country, and the trick was pertinaceously persisted in, so that all the Ophthalmics were left behind; but on the return of the Regiment to Lisbon, I was enabled, having made some discoveries, to convince the Officers of the cowardice and villany of these malingerers. Instead of medicaments, ridicule, shame and punishment were resorted to, and the whole, with the exception of 6 or 7 who had irreparably injured their eyes, were brought back in the course of a few days to the ranks. In about two months thereafter, this Regiment was again ordered to take the field with Sir John Cradock, when the Ophthalmics relapsed; about 100 were presently taken ill. A vigorous system of Drill in heavy marching order, while their Comrades were living at ease in cantonements, was resorted to; they were then shut up, not thro' fear of spreading the infection, but that they might more effectually be deprived of the indulgencies of Wine and animal food, of which the rest had a full ration, and an order was issued that no Ophthalmic, even the totally blind, was on any account to be left behind when the Army marched forward. The Ophthalmia disappeared as if by enchantment and the Regiment remained in the Field perfectly healthy. In the course of the Summer, a few trifling attempts were made to revive the imposture, by putting

pipe clay and urine into the eyes; this took place at Caldas and was detected both in the 31st and 27th Regiments, composed of similar materials, but they were visited by the prompt and disgraceful punishment of flogging on the breech, after which Ophthalmia was no longer heard of in the Army of Portugal and Spain.

From all the foregoing, in opposition to my former preconceived opinions, I am irresistibly led to the conclusion that the whole is an imposture of the most flagitious kind; and if it be true, as I have seen, that the Disease is almost entirely confined to one Class of people (viz; the newly enlisted Irish), that it shows itself amongst that class at particular times and under the suspicious circumstances of embarkations, hard Drills, etc., that the class alluded to have never been in Egypt, in a degree that should entitle them to a preference and predisposition to the Disease before other Soldiers, supposing it to be infectious or contagious, and lastly that the trick has been most completely exposed and detected not only by myself in the 31st Regiment, but by a regularly constituted Court in the 28th Regiment, my opinion may be entitled to some weight.

On other grounds, it may not appear improbable; everyone knows how universally the conspiracy for counterfeiting diseased legs obtained amongst our Soldiers; many more procured their discharge from the successful practice of this deception than have lately done for being blinded by Ophthalmia. The trick of the sore legs came however to be so well understood that it was disgraceful for any Surgeon of a Regiment to be imposed on by it. It therefore, entirely ceased throughout the Army, but on the occasion of our Troops returning from Egypt with numerous invalids from Ophthalmia, the conspiracy revived to new and more successful speculation on this recently imported disease. Let it however be supposed that all my views as here stated are erroneous, and that the disease is real and unfeigned; still I must revert to facts, where, from being an eye witness, I cannot possibly be mistaken, viz: the inefficacy or rather hurtful tendency of confinement, separation and medicaments and the immediate beneficial effects of free exposure to the open air and active exercise. Hence the plan of Dr. Vetch, which goes to perpetuate confinement and to preclude the benefit of free air and extended exercise, does not seem to me to be well adopted to the extermination of Ophthalmia. In other points of view I must also hold it to be objectionable. If the Disease be contagious (yet this contagion has never spread from the Barrack Room of the Soldier to the contagious dwelling of the inhabitant, nor affected in its aggravated form the soldiers of the English Militia, tho' living

under the same roof) we have learnt from the melancholy experience of all great general Hospitals in every service that, to bring an enormous mass of the infected under one roof is not the way to eradicate its seeds, but on the contrary to give invincible activity to its power of destruction. The means of separation are to be had at every Regimental Hospital, and it should depend on the Medical Officer there to enforce them. If infectious, the same rule is applicable, for the means of separation are equally in the Surgeons power, and Patients will have a better chance of being cured, or at least escaping reinoculation at the quarters of their own healthy Regiment than in a community completely contaminated with the infectious disease; or lastly, if it be feigned, an Ophthalmic General Hospital or Depot can serve no purpose that I see but that of a receptacle for conspiracy, where the Villains of the Army may have freer opportunities of maturing their Plans of imposture and rendering detection more impracticable. I would on the contrary propose that the present establishment be broken up, and the patients remanded to their Corps, there to be cured or discharged, but without pensions, under the responsibility of their respective Surgeons. To have ever taken them away unless totally blind under any circumstances (that of foreign service certainly not excepted) I consider to be wrong. It must have acted as a prohibition to investigation, a proclamation of idleness and freedom from responsibility to that class of Medical Officers, who would have had it in their power fully to investigate the Disease, and whose activity might have been depended upon, because their interest and character would both have been at stake. This good effect may still be partially obtained by sending back these patients to their Regiments; and hereafter, in fresh cases, the Commanding Officer may probably prove the best Inspector, the Adjutant the best Surgeon, and the Drummon the most proper Dispenser of cures—the Medical Officer, however, always attending to verify and Warrant the quantum time and fitness of the dose. It is to be lamented, I must farther think, that the proceedings of the Court which sat on the Conspirators of the 28th Regiment and sentenced a considerable number to transportation should have been withheld from the public; had these been promulgated, as they have not even been heard of by a great majority of the Army, every Medical Officer would have been on his guard against the imposture and understood the means of detecting it; its circulation now would in all probability deter others from such attempts, and if read at the Ophthalmic Depot might even shame the more practised adepts of that establishment.

Before I conclude, it belongs to my place in the Medical Department of the Army, and I owe it to my profession, to consider

more fully the pathology of this new Disease, and state my reasons why I pronounce it to be fraudulently and fictitiously made. Everyone knows that Ophthalmia may spread epidemically in this, or any other country, and that the symptoms may run so high as to impair or destroy vision, but there have been hitherto rare and sporadic instances, certainly not exceeding one case in the 100, and never occurred before without some constitutional disturbance, or sympathy from so dreadful a local attack of acute Disease.

We may suppose that local deposition indicative and critical of constitutional indisposition may be formed upon the eye as we see it on other parts of the body in Gout, Piles, Phlegmon, etc., but these last are even preceded or accompanied by strongly marked constitutional disturbance. In these respects, therefore, the present Ophthalmia, which is characteristically free from local pain and general disturbance, differs from all other diseases, and is an exception to the established laws of the animal economy. It has been said to be a new form of contagion imported from Egypt; but where and why did this contagion lie so long dormant (upwards of four years) till it appeared so suddenly on the 28th Regiment, where it was detected to be an imposture?—and why have its ravages been since confined almost exclusively to the newly enlisted Irish, who cannot have held more communication with the Army of Egypt than other Soldiers. Besides, the French who lived in Egypt never heard or dreamt of their endemic Ophthalmia; (if we are to believe Assalini) the produce of the soil and climate of Egypt possessing either an infectious or contagious quality, and I can declare for myself that I never saw an instance of its communication where there was not much occasion to suspect collusion in trick, and I have seen other instances and made experiments, where, if contagion or infection had in any degree existed, it is perfectly impossible, but that it must have been communicated to the healthy but honest subjects who were purposely exposed to its influence.

But on this very interesting point it behoves us to examine still more closely. It is recorded, and I have known a man seized, while on guard, with the new Ophthalmia which would run a course so rapid and dreadful as to destroy the organ of sight within 48 hours or even less; yet under this tremendous visitation the patient's general health was in no way disturbed, the circulation in no degree hurried, nor the appearance of the tongue changed, altho' the whiteness of this last uniformly characterises every inflammatory disease from a Whitloe of the finger to an inflammation of any of the great vital organs. Here then is at once a decided exception to the laws of nature, and a nullification of those hitherto unerring signs and warnings with which we are

endowed by the Wisdom of the Creator for our preservation. How can this have happened?—for such appearances were never before seen, either in the Ophthalmia of this country nor of Egypt. I say with confidence, by the application of some caustic substance to the eye, of sufficient potency to destroy at once the fine nerves of its external coats; those are the seat of its exquisite sensibility, which is great in proportion to the importance of the organ to which they are the outpost and the Guard. Thro' them all natural disease must make its inroads, and the warning which they give to the patient is not to be mistaken; but if these are suddenly destroyed by a potential cautery, and the cord or key, on which the higher ranges of sensation in their connecting series depend, is unstrung or demolished, the whole organ, being thus put without the pale of the constitution, tumbles at once into anarchy and annihilation, before the unopposed action of the artificial agent.

To illustrate this, and that I may not be suspected of using wild theoretical language, I shall take a familiar instance from the improved surgery of the present day. In the guarded application of a caustic to the inner surface of the urethra for a few seconds, we have an illustration of what must happen to the eye, when the same substance is used for hours together to produce the appearance of disease. The nerves of the strictured urethra are endued with an acute and morbid sensibility that can scarcely bear the contact of a blunt innoxious instrument, and thro' them diseased impressions are conveyed which more or less affect the general frame; yet the application of a caustic gives only a momentary pain, and by destroying the small nerves which are the seat of sensation and conductors of irritation never failingly gives a respite to the present symptoms of the disease, and deadens the future sensibility of the passage.

To indulge this disquisition further would be foreign to the purpose. But before I conclude, I wish to vindicate myself from even the appearance of illiberality on two points of the foregoing, when, to do justice to my subject, I was obliged to give free licence to my pen. In speaking of the Irish Soldiers as being commonly the Manufacturers of spurious Ophthalmia, I need only in explanation advert to the politically unsettled state of that country inducing habits of mind little favourable to the principles of steady and regular service; and when I talked of discharging the blind without pensions, I could not presume to throw any general imputation on that most wise and benevolent regulation, for I should be worse than blind myself if I did not see that if the service were converted into a Slavery, without end and without recompense, a better and more virtuous description of men would in all probability be induced to conspire the means of redemption

than the present Ophthalmics. I may believe in the conspiracy even if I had not particular and positive proofs, because I see that men desert notwithstanding all the risks and penalties. It is not therefore to be wondered at, that many of them would run the risk of blindness to effect their emancipation and obtain a pension for life besides. After all, tho' I cannot be mistaken with regard to what I have actually seen and detected in different services, the present patients at the Ophthalmic depot may be of a very different description from those I have been treating; I wish therefore that my opinion thus officially given may invite further enquiry, so that the fullest elucidation may be obtained. Dr. Vetch's opportunities of investigation have been infinitely superior to mine, and from his acknowledged zeal, talents and impartiality, there cannot be a doubt he has fully used them. I do not therefore pretend to give the history of his patients, but only of my own. From his writings I have reaped both satisfaction and instruction, and in whatever relates to the true pathology of the eye I freely acknowledge him my Master.

I have the honour to be with the greatest respect

Sir,

Your most obedient and humble servant,

(Signed) Wm. Fergusson.

To

Francis Knight Esq.,

Inspector General. Etc. Etc. Etc.

What a very illuminating sidelight do the contents of this letter throw upon army life, and the incidence of ophthalmia in the British Army, one hundred and thirty years ago. More than that, it is pleasant to ponder over the revelations of the mentalities of the writer of the letter and of the surgeon whom he is criticising. One cannot but be struck, on perusing the writings of Staff Surgeon Vetch, with the thoughtful dignified approach to his subject which he always adopts. One appreciates that one is dealing with medicine well over a century old; none the less here, manifestly, is a quiet-minded scientific gentleman pursuing his subject in sincerity, treating his patients with kindness, and answering his critics—and they were many—with reasoned argument and dignified reproof. It is impossible not to contrast the attitude of Dr. Fergusson with that of Staff Surgeon Vetch, for in the former's letter the dominant note is one of truculent superiority, made all the more obvious by his unctuous apologies, and his insincere admission of the greater experience and knowledge of the surgeon whom he is criticising. That his thesis was

the outcome of his experience one has no right to doubt, and it may well be that factitious ophthalmia was prevalent at the time at which he writes; but it is absurd on his part to suggest that his rough and ready methods would constitute adequate treatment for the military pandemic which Staff Surgeon Vetch was endeavouring to combat. Vetch may have been deceived on occasions; Fergusson may have met more than his share of artefacts, but one cannot but feel that he shows himself to be the bluff, blustering regimental officer, and that the broad generalisations and drastic suggestions of this letter compare unfavourably with the thoughtful utterances of the man he criticises.

So much for that side of the question. If it is considered that it has been dwelt upon at too great a length, and that it is nothing more than a digression, the writer apologises, and pleads in extenuation that it was this letter, and the side of the question which it represents, which turned his attention particularly to the subject of the nature of " Egyptian Ophthalmia " and prompted the writing of this article. Let us return to the main subject.

Those who hold that it was not trachoma which affected the troops in the Napoleonic campaign base their opinion mainly upon two considerations: firstly, that the disease and its sequelae as described at the time differ fundamentally from trachoma as we know it to-day, and secondly, that the epidemic of trachoma in Europe followed some months or even years after the return of the armies to their native countries. Neither of these considerations can be said really to back their opinion—or at any rate to disprove the opposite contention.

The clinical objection is the weaker of the two. Trachoma is in its early stages quite typically complicated by inflammatory manifestations, even in this country and at this time; it is probable that such complication was even more likely one hundred and forty years ago. Larrey, Napoleon's surgeon-in-chief on the Egyptian campaign, is often quoted in an attempt to disprove the thesis that the troops were affected by trachoma; the following is the relevant passage:

" Swelling of the lids and of the conjunctiva, and sometimes of the coats of the eye; extreme local pain, attributed by the patient to the presence of grains of sand (these are dilated vessels); diminution of vision and inability to stand a bright light. To these first symptoms soon succeeded violent headaches, giddiness and insomnia. The few tears which are secreted are bitter and irritate the lids and puncta lachrymalia. All these symptoms are aggravated and are frequently followed by fever, sometimes even by delirium. The disease reaches its crisis on the third or fourth day, sooner with some individuals, later with others. The

termination varies. When it is inflammatory and is left solely to the resources of nature, there form ordinarily towards the sixth or seventh day points of suppuration on the edges of the lids, on the external surface and at the angles. The ulcers spread by degrees on to the conjunctiva, attack the cornea and often perforate it. Sometimes the cornea gives way suddenly, without ulceration; I have seen several such cases. The rupture occurred within the first 24 hours, when the conjunctiva was hardly red, and it is difficult to understand the cause of this rapid and spontaneous rupture. We content ourselves with observing the phenomena which have occurred in Egypt, and the effects which have been produced. The opening which resulted is round and of a diameter almost the same in all the patients who have been attacked. It allows the passage of a portion of Descemet's membrane or of the iris, and forms a hernia, known by the name of staphyloma. The swelling formed by Descemet's membrane is a dull grey (*gris terne*), that of the iris of a darker colour. This swelling is painful to the lightest touch of outside objects, and to the rubbing of the lids. The vision during the early days is more or less diminished, according as the pupil is partly or entirely obscured; but generally the staphyloma shrinks by degrees, goes back into the anterior chamber, and the membranes resume their previous positions. Sometimes there remains a small portion outside, which is strangled by the closing of the aperture, loses its sensitiveness, and acquires a certain toughness, or else it swells and divides into several globules and takes on a carcinomatous character, especially if there is any complication of venereal disease."

His further description of the shrunken globes which followed supports the opinion that a purulent ophthalmia was the cause; his statement that the right eye was affected more often than the left, and that nearly all lost eyes were right eyes (though this occurrence was attributed by him to the fact that since most men sleep on their right side the humidity of the earth affected the right side more) would appear to indicate an infection of gonorrhoeal origin. None of these facts disproves the contention that the troops were infected with trachoma at the same time; nor by any means does Larrey's specific statement that "the tarsal cartilages were rarely affected," for it is extremely unlikely that this manifestation of trachoma would be evident so short a time after infection. The fact that Larrey mentioned the "tarsal cartilages" at all is of interest in that it indicates that the possibility of trachoma was present in his mind.

The clinical objection would therefore appear to be far from insuperable. The second objection may be countered with equal weight. If trachoma breaks out in a community it is a matter of common knowledge that, on making a routine examination,

one may find numbers of the community to be suffering from well-established trachoma which has caused little or no discomfort to the affected individuals. Again, other members of the community may have admittedly suffered from an acute ophthalmia which, in spite of their subsequent belief that they were rid of all their eye trouble, has been the starting point of a chronic trachomatous infection which has not made itself evident until after a latent period of maybe considerable duration. Therefore one may fairly assume that, although there were numerous cases of trachoma in the armies while on campaign, and also immediately afterwards (and this is admitted on all hands) there is nothing inconsistent with our experience of the disease in the fact that a generalised outbreak of trachoma, caused by infection of one soldier from another, should be delayed until some months or years after the return of the armies to their native lands. Many other factors might combine to determine the time of onset of this outbreak, the sole necessary factor being an existing nucleus of infected units in the armies.

There is no doubt that trachoma was known in most, if not all, of the countries concerned, before the time of the Napoleonic campaign. That the troops were also heavily infected with gonorrhoeal ophthalmia is also fairly evident—and in this connection one must similarly consider the fact that the manifestations of the Egyptian form of this disease may differ profoundly from those which we are accustomed to seeing in this country. The coccus is in a modified state and behaves in a very different manner in the two countries; the clinical course in Egypt may be much less acute, and transmission appears to take place in a fashion similar to that of a Koch-Weeks' infection. Indeed, the disease may almost be described as " non-venereal " in nature. Similarly, as Fuchs says, trachoma has probably changed its nature profoundly in the course of time, and this may well be, in part, the cause of the diversity of opinion upon the subject.

To one, therefore, who, while not pretending for one moment to be anything in the nature of an authority on the question, has at least given the matter some particular attention, it has become apparent that the Napoleonic campaign may fairly be blamed for the epidemic nature of trachoma in Europe in the years immediately following the campaign. Factitious ophthalmia may have been rife, as Fergusson declares; the original infection may have been complicated by a purulent ophthalmia; there is no doubt that trachoma was well known in Western Europe before the Egyptian Campaign, nor that the Irish recruits were heavily infected at the time of their enlistment; none the less a consideration of the history of the disease leads one to the conclusion that it was from Egypt that the infection was brought

which was to prove in later years such a wide-spread and sight-destroying scourge.

REFERENCES

BOLDT.—"Trachoma." Translated by J. H. Parsons and T. Snowball. London. 1914.
COLLINS.—Introduction to Boldt (above).
CUÉNOD ET NATAF.—"Le Trachome," 1930.
DUKE-ELDER.—Text-book of Ophthalmology. Vol. II. London. 1938.
EASON.—(1) *Trans. Ophthal. Soc. U.K.*, p. 38, 1918. (2) *Guy's Hospital Reports*, Vol. LXX, p. 63.
EBLE.—"Die . . . agyptische Augenentzundung." Stuttgart. 1839.
FUCHS (DUANE).—Text-book of Ophthalmology. Eighth edition. 1924.
LARREY.—"Memories de Chirurgie Militaire et Campagnes." Paris. 1812.
MACCALLAN.—"Trachoma." London, 1936, and private communication.
MEYERHOF.—*Brit. Jl. Ophthal*, p. 129, 1932.
VETCH.—(1) "A Practical Treatise on the Diseases of the Eye." London. 1820. (2) "An Account of the Ophthalmia which has appeared in England since the return of the British Army from Egypt. London. 1807. (3) Observations relative to the Treatment by Sir William Adams of the Ophthalmic Cases of the Army." London. 1818.

OCULAR CHANGES ASSOCIATED WITH NAEVUS FLAMMEUS

BY

J. JAMESON EVANS *and* P. JAMESON EVANS

BIRMINGHAM

THE influence of maldevelopment of the orbital vessels upon the optic nerve has been widely discussed in recent years. Many types of malformation, localised to various sites in the globe and orbit, have been described, and their existence in association with intra-cranial and facial lesions noted (notably by Lindau,[9] Lawford,[8] Cushing[3] and Knapp[6]). The question of whether optic atrophy, with cupping, so often found in such cases, is the result of true glaucoma with tension or, maybe, a primary atrophy due to a disturbance of the nutrition of the nerve, and in which the intra-ocular tension is not, of itself, a factor of importance, is still undecided. It may be recalled that Schnabel[13] strongly advocated the view that, even in true glaucoma with increased tension, the cupping of the optic disc was independent of the state of the intra-ocular tension and was due to an essential lacunar atrophy of the optic nerve. In association with haemangiomatosis, and other malformations of the vessels of the eye and orbit, Löwenstein,[10] Bär,[1] McRae,[11] and O'Brien and Porter[12] report instances where there has been no demonstrable rise of tension: on the other hand the majority of cases of this type of lesion have been associated with an increased intra-ocular tension and, bearing in mind

that the volume of the intra-ocular contents in such cases is especially subject to sudden increase, this is only to be expected.

Further, such malformation, affecting the vascular coat of the eye, is often found to be associated with other changes in this layer (abnormal development of the filtration angle, etc.), so that abnormalities of tension are to be expected. Indeed, it would appear that little more proof is available that increase of tension results in optic atrophy than that the reverse process is true. Thiel[15] has recently drawn attention to instances of optic atrophy with cupping, indistinguishable from that of chronic primary glaucoma, but without tension, in which sclerosis of the internal carotid arteries is held to be the basic factor. Knapp[6] also has published a series of cases of this type in which sclerosis of the basal cerebral vessels was a constant finding.

In the present paper two cases are submitted for consideration: the one showing increase of tension, the other with normal tension over a number of years of observation. Both are instances of naevus flammeus with associated ocular changes.

Case 1. P.R.E., aged 23 years. When first observed in 1931, presented an extensive naevus flammeus of the whole face extending up to the hair line and some distance down the neck. The mouth and palate were also involved; the reason for wishing an ophthalmic examination was recurrent inflammation of the left eye. This was of the nature of a patch of superficial vascular keratitis in the upper and outer quadrant of the cornea, the resultant nebulae involving a part of the pupillary area.

The intra-ocular findings in this eye, as also in the right eye in which the cornea was normal, were a pronounced development of reddish tortuous vessels in the iris stroma which appeared slightly thickened, and of the circulus minor, and of bunches of tortuous vessels in the neighbourhood of the filtration angle. The media were clear and there was no suggestion of opacities in the lens or vitreous. The pupil reflexes were normal; the anterior chamber was of normal depth in each eye. The optic discs were deeply cupped, about three-fourths of the discs being involved, and the cupped area showed distinct pallor. The retinal vessels were not definitely abnormal in size, but they were rather tortuous. Vision was normal in the right eye, but in the left vision was only 6/24 owing to corneal opacity and irregularity of surface. The fields showed temporal contraction to 20°—30°, more marked in the left field. There was, during observation, no variation of intra-ocular tension, which was normal, and at no time was an increase of tension noted, or the probability of it suggested by symptoms or observation. Schiötz 18 mm. Hg in each eye.

The attacks of keratitis in the left eye recurred once or twice a year and the duration of the attacks was generally about fourteen

days. They were associated with some increase of vascularity of the nasal mucous membrane, and there were indications of left antral infection in February, 1934.

During 1934 an attempt was made to cure the facial naevus by means of massive doses of radium by surface applications to the neck and radon needles in the lips. In 1935 the patient complained of impairment of vision in both eyes. At this time no material change was noticeable in the external appearance of the eyes, but there was slight but definite fibrosis, with some decoloration, of the facial angioma. The lips, however, became more congested and oedematous, showing a tendency to partial necrosis. The dilated and tortuous vessels in the iris were less obvious and showed definite sclerosis. The pupil reflexes were still normal. X-rays of the skull at this time showed enlarged and tortuous vessels of the meninges and a circular calcareous area in the occipital pole of the brain. The lenses showed the posterior cortical cataract, saucer-like in type, commonly seen in glass-blowers and known as ray cataract. The other media and the fundi were unchanged. Vision—R.E.=6/18, L.E.=6/36. The use of atropine to improve vision had no effect on the tension which remained unchanged. The cataract was regarded as being the result of the heavy radium therapy. This view is supported by Mr. Harrison Butler and Mr. Stallard, whose observations are appended. At present, the lenticular opacities appear to be stationary. The facial deformity is now being treated by plastic surgery.

Case 2. A.C., aged 12 years. Attended the Birmingham and Midland Eye Hospital with pain in the left eye of three weeks' duration, and the history that the eye had been blind since birth and that it had previously given pain on a number of occasions. Right Eye—vision 6/6, this eye was normal in every respect. Left Eye—vision, no perception of light. Tension 42 mm. Hg Schiötz. There was congestion of the conjunctival and anterior ciliary vessels. The cornea was oedematous; the anterior chamber shallow, with a small hyphaema below: the iris vessels prominently coursing over the anterior surface. The lens showed a diffuse intumescent cataract. Transillumination gave an even but rather dim reflex.

The face showed a left sided naevus flammeus, confined to the approximate limits of the skin distribution of the maxillary division of the fifth nerve, but overlapping the ophthalmic area a little. The mucous membranes of the cheek, palate and soft palate on this side were involved.

Other parts of the body presented areas of skin haemangioma. A full neurological investigation showed no evidence of lesions involving the central nervous system. X-rays of the skull showed no areas of abnormal calcification, but the left optic foramen was slightly larger than the right.

A provisional diagnosis was made of secondary glaucoma due to intra-ocular haemorrhage resulting from the presence of haemangioma of the choroid.

The eye was removed the next day, without excessive bleeding, and the diagnosis was confirmed by pathological examination.

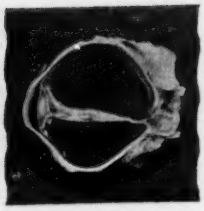

FIG. 1 (*a*).

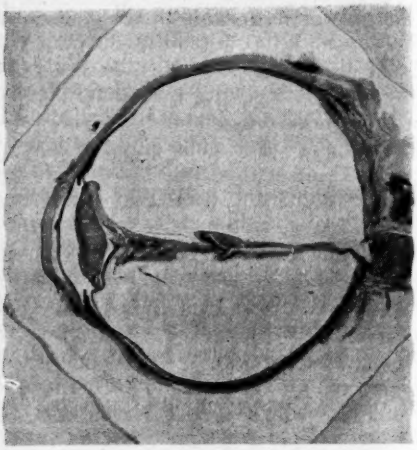

FIG. 1 (*b*).

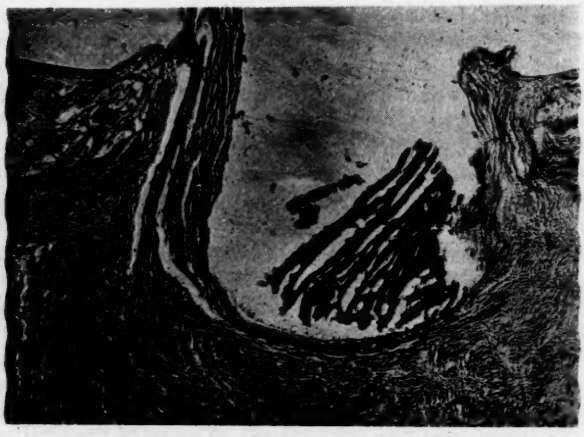

FIG. 2.

The microscopic study of the eye revealed a flat growth of haemangiomatous tissue of the choroid occupying the posterior third of the globe's circumference: the retina was completely detached and the sub-retinal space occupied by recent haemorrhage. No vitreous existed, the detached retina forming a double-layered fold in the centre of the globe (Fig. 1). The optic disc was deeply cupped (Fig. 2) and the nerve greatly atrophied and

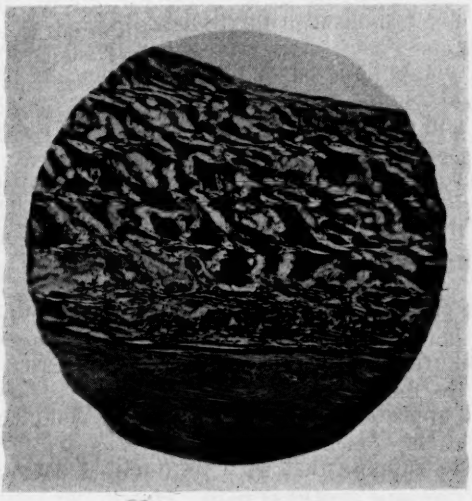

FIG. 3.

FIG. 4.

presenting, in addition to small haemorrhages, many spaces of lacunar atrophy. The filtration angle of the anterior chamber was quite closed by the forward displacement of the iris and lens. The growth itself consisted of a flat wide thickening of the choroid occupying the posterior pole of the globe, very similar to that described by Jahnke[5]; the enormous distension of the vessels is easily seen in the sections under the microscope (Fig. 3). We

were particularly fortunate in being able to demonstrate the course of the communication of the intra-ocular tumour through the sclera to the extra-ocular veins, the track following the path of the vorticose vein (Fig. 4). This suggests that the growth itself is essentially derived from the venous side of the vascular tree and not from the arterial, an observation which may have some bearing on the co-existence, in some cases, of areas showing calcareous degeneration in the occipital lobe and elsewhere. Recent work on venous stasis has emphasised its important bearing on the failure of metabolism in the tissues.

This case has already been reported in the American Archives of Ophthalmology (August, 1937) as an instance of haemangioma of the choroid (but the question of optic atrophy was not then fully discussed), and is described again by courtesy of the editor of that journal.

In respect of the second case (A.C.) the optic atrophy is very advanced and the cupping deep. What is the mechanism of the cupping of this case? That there was a raised intra-ocular tension on several occasions may be presumed by the history of recurrent attacks of pain extending over a number of years. That which brought the patient to our notice was apparently similar to its predecessors, though more severe in degree. The diagnosis of intra-ocular haemorrhage being the cause of the increased tension was confirmed by the findings in the excised eye, and, from the degenerated appearance of the retina and its completely detached condition, leaving practically no true vitreous space (Fig. 1). It is reasonable to suppose that such retro-retinal haemorrhages had caused the previous attacks of pain. Hence, one cannot hold that increased pressure in the vitreous space had produced cupping of the disc directly. Moreover, the detached retina is not pressed down into the disc although it lies directly in front, being firmly attached at the disc margin, so that the pressure from retro-retinal haemorrhage would not appear to be responsible. It is suggested then that the cupping is the result of primary atrophy of the nerve consequent upon impaired nutrition, associated with the abnormal state of the blood vessels supplying the nerve. The histology of the nerve also shows a comparatively loose structure, in no way conveying the impression of compression, and in which are found, not only spaces filled with blood, but others suggestive of lacunar atrophy. However, since this case has been complicated by repeated attacks of raised intra-ocular tension the thesis is not indisputable.

Turning, therefore, to the first case in which there is deep cupping, with pallor of the discs; increased tension has never been noted during a period of seven years under observation, nor have there ever been symptoms which might suggest increased

intra-ocular pressure. In fact the tonometric readings have never reached 20 mm. Hg (Schiötz); and up to the time of the unfortunate formation of (radium) cataract there had been no appreciable alteration in visual fields or acuity.

In Knapp's[7] report of optic atrophy with cupping, associated with sclerosis of the basal cerebral vessels, the average age of the patients is 65 years. While some degree of senile change of vessels is to be expected at such an age, the changes are not usually sufficiently advanced to be demonstrable by plain X-ray photographs of the skull. In many cases of naevus flammeus the sclerotic and degenerative changes (frequently with some amount of calcification) are so marked as to be easily demonstrable by X-ray in young patients, whilst in those cases showing homonymous hemianopic field changes (D. Campbell,[2] H. Tyson[16]) as a result of the presence of a haemangioma in the occipital lobe of the brain, there are obvious patches of calcareous degeneration.

Cushing and Bailey[4] have commented upon the tendency of venous angiomata to undergo calcification. The incidence of occipital angiomata in these cases appears to be more than fortuitous, but the governing factors are at present entirely unknown.

Thiel[15] also has published a number of cases in which atrophy and cupping of the optic disc, indistinguishable from that of glaucoma, has occurred in association with sclerosis of the internal carotid vessels and without increase of the intra-ocular pressure, he has further demonstrated the presence of nasal field defects in such cases.

In the second case (A.C.) no sclerosis of intra-cranial vessels was observed, but in the first (P.R.E.) there is marked opacity of the internal carotid and middle meningeal vessels and, in addition, a sharply defined calcareous area in the occipital region (but without any hemianopia in the fields). This sclerosis constitutes a sign of defective nutrition of the vessel walls and provides evidence also in favour of a similar defect in the nutrition of the tissues supplied by these vessels—among which is included the optic nerve.

It might be expected that, in such cases, the lens, dependent as it is for its transparency on adequate nourishment from the surrounding fluids and hence, indirectly, on the state of the vessels of the uveal tract, would be affected and that cataract would be commonly found. This, however, does not appear to be the case either in Knapp's series of ten cases or in those cases of haemangioma retinae described by Lindau. In other cases the incidence of cataract is complicated by the presence of buphthalmos, etc.

Hence, in the second case the formation of cataract may have been due to the sudden increase of tension and to its effect on the passage of oxygen through the lens capsule. Evidence in favour of this is found in the intumescent and diffuse character of the cataract, giving the impression of recent development.

In the first case the cataract is of a very different nature. When first seen this case presented no opacity of the lens in either eye, the opacities developing some two months after the institution of radium therapy (1934) to the face and lips with which their incidence appears to be closely related.

FIG. 5 (*a*)

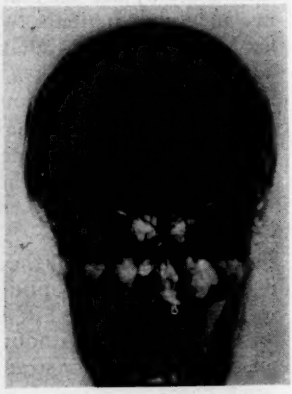

FIG. 5 (*b*)

Reduced X-ray diagrams of skull of P.R.E.
(*a*) Lateral view. (*b*) Antero-posterior view.

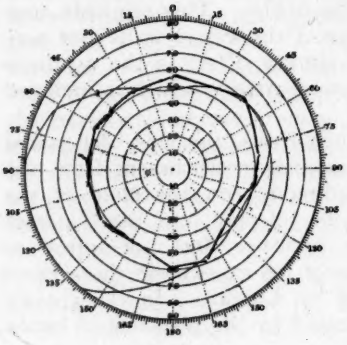

FIG. 6 (*a*)

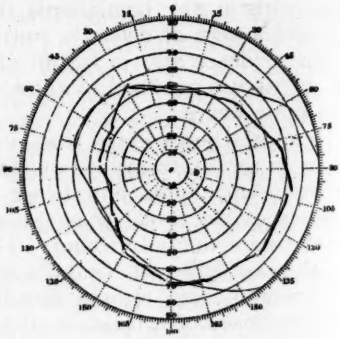

FIG. 6 (*b*)

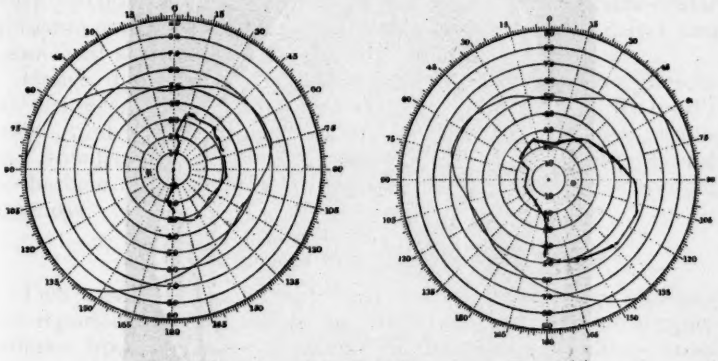

FIG 6. (*c*) July 27, 1938. FIG. 6 (*d*)

P.R.E. Fields of Vision ($\frac{1}{2}$° white).

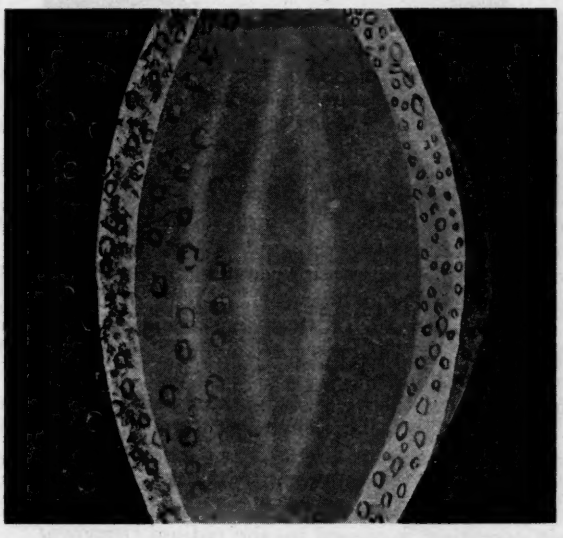

FIG. 7 (*a*)

Drawing of the lens seen with the broad beam of the slit-lamp: showing the split appearance of the posterior capsule with bulging of the posterior layer giving the appearance of a lenticonus posterior: also the vacuolation.
(By courtesy of Mr. T. Harrison Butler.)

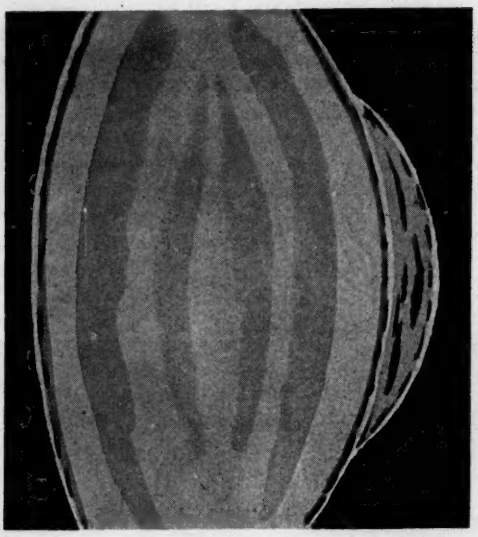

FIG. 7 (*b*)

Drawing of the same lens seen with the narrow beam of the slit-lamp. (By courtesy of Mr. T. Harrison Butler.)

We are indebted to Mr. Harrison Butler for comments upon the slit-lamp appearances of the lens. " Under each anterior capsule there are a number of large vacuoles. The anterior lens shagreen is more easily seen, and is of a coarser type than in the normal lens. The posterior capsule appears to be split, giving an appearance similar to that seen in lenticonus posterior. The cortex in front of each of these posterior layers of capsule is vacuolated, but the vacuoles are smaller than those seen in the anterior part of the lens.

" The lens matter between the split capsules is also vacuolated. There is a double vacuolated layer (Figs. 7(*a*) and 7(*b*)). The appearances correspond closely with those of X-ray cataract described by Vogt[17, 18] but in this case the posterior bulge and double layer are more pronounced. There is no doubt whatsoever that this condition is caused by the radium treatment."

Further, Mr. H. B. Stallard, who gave an opinion on this case, also considered the lens opacities to be the result of the radium therapy. He writes that " the lens changes in the posterior layers of the cortex are typical of irradiation cataract and have probably been caused by the heavy dosage of radium two years

ago." This authority further remarks "the optic discs are deeply cupped but, in the absence of increased intra-ocular pressure and field loss, I presume this is a congenital defect and associated with intra-ocular vascular abnormalities."

Hence, it seems fairly certain that this case does, in fact, present an instance, of which other examples are quoted in Mr. Stallard's monograph on the subject[14] of delayed cataract formation resulting from irradiation. Such cases are rare, but perhaps are destined to become more common with the greater frequency of radium therapy.

Summary and Conclusions

Two cases associated with optic atrophy and other changes are reported of naevus flammeus. It is held that the optic atrophy follows upon defective nutrition of the nerve resulting from abnormal vascular supply, and is not secondary to, nor dependent upon, a rise of intra-ocular tension.

In the one case there was increased intra-ocular tension on at least one, and probably on several, occasions during a long history; in the other case no increase of tension has been observed during a period of thirteen years. It is held that, certainly in the latter case, and possibly in the former also, optic atrophy is independent of changes in the intra-ocular pressure.

The second case presents the rare opportunity of microscopic examination of a haemangioma of the choroid and particularly of the course of entry of the haemangiomatous tissue into the globe. The first case shows the presence of associated intracranial vascular abnormalities and of irradiation cataract.

BIBLIOGRAPHY

1. BAR, C.—*Zeitschr. f. Augenheilk.*, Vol. LVII, p. 628, 1925.
2. CAMPBELL, D. C.—Personal communications, 1937.
3. CUSHING, H.—*Jl. Amer. Med. Assoc.*, Vol. XLVII, p 178, 1906.
4. CUSHING, H. and BAILEY, P.—Springfield, Illinois (C. Thomas), 1928.
5. JAHNKE, W.—*Zeitschr. f. Augenheilk.*, Vol. LXXIII, p. 219, 1931.
6. KNAPP, A.—*Arch of Ophthal.*, Vol. LVII, p. 219, 1928.
7. *Idem.*—*Arch. of Ophthal.*, Vol. LXV, p. 637, 1932.
8. LAWFORD, J. B—*Trans. Ophthal. Soc. U.K.*, Vol. V, p. 136, 1885.
9. LINDAU, A.—*Acta Ophthal.*, p. 193, 1927.
10. LÖWENSTEIN, A.—*Klin. Monatsbl. f. Augenheilk.*, Vol. LXX, p. 540, 1923.
11. MCRAE, A.—*Brit. Jl. Ophthal.*, Vol. XIII, p. 63, 1929.
12. O'BRIEN and PORTER.—*Arch. of Ophthal.*, Vol. LXVI, p. 715, 1933.
13. SCHNABEL.—*Arch. f. Augenheilk.*, Vol. XXIV, p. 273, 1892.
14. STALLARD, H. B.—Radiant Energy. Monograph Supplement, *Brit. Jl. of Ophthal.*, 1933.
15. THIEL, P. J.—Röntgendiagnostik des Schädels bei Erkrankungen: des Auges und seiner Nachbarorganen. Pp. 134-143. 1932. (Julius Springer, Berlin).
16. TYSON, H. H.—*Arch. of Ophthal.*, Vol. VIII, p. 365, 1932.
17. VOGT, A.—Spaltlampenmicroscopie. Vol. II, p. 709. 1931.
18. *Idem.*—Spaltlampenmicroscopie. Vol. II, p. 712-714. 1931.

THE FLUID EQUILIBRIUM OF THE BODY AND ITS RELATION TO THE EYE*

BY

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(FROM THE COURTAULD INSTITUTE OF BIOCHEMISTRY, MIDDLESEX HOSPITAL, W.1)

IN various sites throughout the body, fluids of different chemical composition are produced and these include saliva, gastric juice, pancreatic juice, bile, urine, aqueous humour, cerebro-spinal fluid, tissue fluid and lymph. The common origin or mother liquor of all these fluids is the blood, although the mechanism of formation of these diverse biological products is not similar. Of these various fluids, tissue fluid and lymph would appear to be closely akin to blood plasma and a well-marked inter-relationship has been drawn between the three fluid systems to which they belong—namely, the connective tissue or cellular interspaces, the lymphatic system and the vascular system.

The Vascular System.—This system contains blood corpuscles and plasma. It is a closed system of tubes leading from the heart by arteries, arterioles, and capillaries to the tissue spaces throughout the body, thence by the veins back to the heart.

The Lymphatic System.—This system contains protein-rich fluid called lymph. The lymphatic system is derived from veins and the lymphatic vessels invade the body as do the blood vessels. These lymph vessels have the same relationship to the tissue spaces as have the blood capillaries. There is thus no continuity between the tissue spaces and the lymph channels.

The Tissue Spaces.—These are intermediate in position between the vascular system and the lymphatic system. This system is by virtue of its structure, function, and contents quite independent of the two preceding systems. The tissue spaces contain fluid and this fluid must be distinguished from lymph. It is important to distinguish between the contents of these fluid systems, for much confusion exists because of the loose way in which people refer to them.

1. *Lymph*: this is fluid inside the lymphatic system.
2. *Tissue fluid*: this is fluid in the areas outside the blood and lymph capillaries in cellular or connective tissue interspaces.

*Lecture delivered at the Royal London Ophthalmic Hospital on November 11, 1938.

3. *Plasma*: this is the unclotted fluid of the blood in the vascular system.

These fluids differ from one another, for example chemically such as in their protein and NaCl content. The protein is greatest in the plasma and least in the tissue fluid.

The nature of the interchange that occurs between these systems is a complex one including the passage of gases, water, inorganic

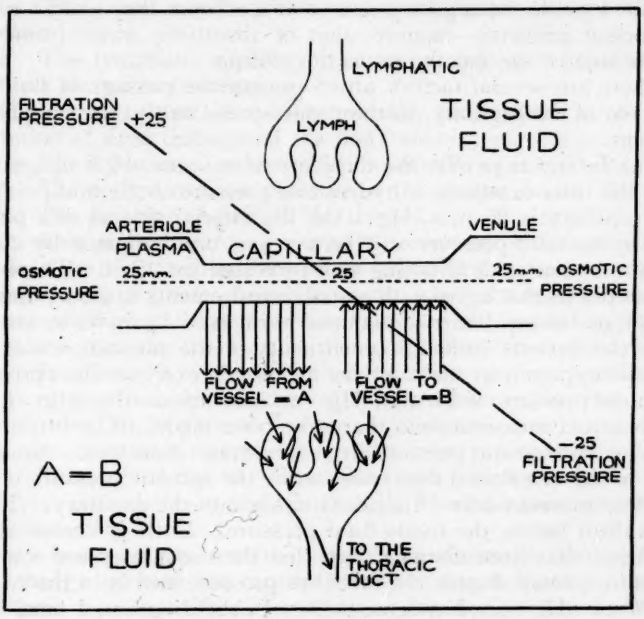

FIG. 1.

salts, organic crystalloids, and in certain tissues colloids. The factors that play a part in bringing about the interchange can best be demonstrated by Fig. 1.

This figure shows diagrammatically the structure of a normally functioning vascular and lymph capillary. A vascular capillary has the following characteristics:—

1. It has an afferent and an efferent vessel. The former is usually an arteriole and the latter a vein but this is not always the case. For example, in the liver, the afferent vessel is a vein not an artery.
2. Its walls are composed of a single layer of endothelial cells.
3. All interchanges of fluid, salts, dissolved gases, take place

exclusively through the capillary wall and not through the walls of arteries or of veins.

4. It has a reciprocal permeability:—*i.e.*, it allows the passage of fluids, salts, and gases across its walls with equal freedom in both directions.

5. Its membrane or wall is semi-permeable; i.e., whereas water, salts, and gases are allowed free passage over the membrane to the tissues, the plasma proteins are not. The plasma proteins are retained inside the capillary and on this account they exert a most important property—namely, that of absorbing water from the tissue side of the membrane to the plasma.

There are several factors which govern the passage of fluid in and out of the capillary, to the tissue spaces and to the lymphatic system.

The Interchange over the Capillary Membrane.—Let us assume that the intra-capillary or hydrostatic pressure at the mid-point of the capillary is 25 mm. Hg. On the arterial side of this point, the hydrostatic pressure will be a rising one indicated by 25+; on the venous side a falling one indicated by 25-. These are assumptions that agree with actual measurements made by Landis (1930) on the capillaries of man and animals. Again let us assume that the various colloidal constituents of the plasma, which are unable to pass over the capillary membrane exert an absorptive or osmotic pressure of 25 mm. Hg—an assumption that also agrees with actual measurements that have been made. The intracapillary or hydrostatic pressure drives fluid and dissolved substances out of the vessel into the tissues while the osmotic pressure of the plasma proteins draws fluid back again into the capillary. There is a third factor, the tissue fluid pressure. Little is known about it, but it has been demonstrated that the tissue pressure counteracts in a small degree the filtration pressure and is in the region of 9 mm. Hg—see Landerer (1884). In the diagram, I have indicated by arrows the flow in and out of the capillary. When as happens normally these forces balance one another, there is no accumulation of fluid in the tissue spaces and there is a constant and continual interchange of fluid occurring.

The Flow of Fluid into the Lymphatic Vessels.—It is probable, though by no means certain, that two forces are at work in guiding fluid into the lymphatic system. These are the osmotic pressure of the lymph, and the hydrostatic pressure of the fluid in the connective tissue spaces. Once the fluid has entered the lymphatic system and becomes lymph, its passage onwards to the large lymph channels and to the thoracic duct is hastened by massage and active muscular movement. There is no doubt that the lymphatic route is the one way that protein is removed from the subcutaneous tissues. The wall of the lymph capillary is much

more permeable to protein than the blood capillary, and Drinker believes that " protein-containing fluid leaving the blood stream moves into the lymphatic almost as easily as through the tissue spaces." (Drinker and Field, 1933). The function of the lymphatics is therefore to remove material from the tissue spaces which is not absorbed by the blood capillaries.

Very briefly then, the relative values of the blood capillary and the lymphatic capillary regarding fluid interchange are as follows:

1. The blood capillary determines the water content of the tissues.
2. The lymphatic capillary removes protein and has little to do with the water content.

The factors, therefore, which normally play a part in the distribution of fluid throughout the body are:—

1. The hydrostatic pressure in the capillaries.
2. The colloid osmotic pressure of the plasma.
3. The pressure of fluid in the tissues.

Do these physical forces govern the formation of the specialised fluids in the body such as the cerebro-spinal fluid, aqueous humour, gastric juice, or bile? In other words, when the mechanism for the interchange of tissue fluid breaks down and water-logging or dehydration of the tissues take place, are these specialised fluids similarly affected? The behaviour of these various specialised fluids throughout the body, after disturbances in the fluid equilibrium, might give an indication as to their nature; whether they are dialysates, secretions, exudates or transudates. It is generally accepted that the tissue fluid and lymph are formed by the process of dialysis, the simple laws of which have just been described.

I am now going to consider conditions where the normal fluid interchange between the capillary and the tissue spaces is upset, and later we shall determine whether the equilibrium in these specialised fluid systems is in any way disturbed. A disturbance in the tissue fluid equilibrium occurs in the following conditions.

1. When the colloid osmotic pressure of the plasma falls.
2. When the osmotic pressure of the blood is suddenly raised.

In the first condition, water-logging occurs, and in the second, dehydration.

A fall in the colloid osmotic pressure of the plasma.—If the osmotic pressure of the plasma proteins falls from a normal of 25 mm. Hg to 10 mm. Hg, a condition represented in the second diagram, is created.

Owing to the fall in the suction power, as it were, of the plasma proteins, there is a relative increase in the filtration pressure. As the colloid osmotic pressure is now no longer able to counterbalance the hydrostatic pressure in the capillaries on theoretical

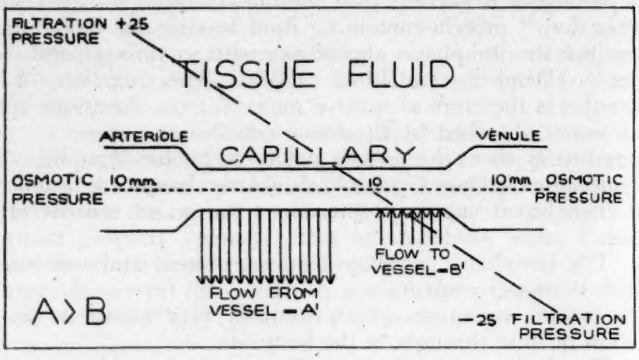

FIG. 2.

grounds, stagnation of fluid in the tissue spaces should result—and clinically in fact this does occur.

The causal relationship between nephrotic or generalised oedema and depleted plasma proteins was first drawn attention to by Epstein (1914, 1917, 1917*a*, 1922), who was investigating the mechanism of oedema in nephrosis. Nephrosis is a disease which is characterised by certain well-marked features—Bennett, Dodds and Robertson (1931).

Clinically.—There is generalised oedema with no cardiovascular changes (thus no retinal changes, no elevated blood-pressure, and no cardiac hypertrophy are present).

Biochemically.

1. Heavy albuminuria.
2. Absence of haematuria.
3. Doubly refractile lipoids in the urine.
4. Oliguria.
5. Reduced B.M.R.
6. The blood chemistry is normal except the plasma proteins are greatly diminished and the cholesterol is raised.

In explaining this syndrome, Epstein has stated that as a result of the massive albuminuria the plasma proteins became depleted (*i.e.*, the plasma proteins were simply lost through the kidneys). A stage was finally reached when the plasma proteins were so depleted that their osmotic pressure was no longer able to counterbalance the hydrostatic pressure in the capillaries which forced the fluid from the blood stream into the tissues. Thus more fluid was pushed out into the tissues than was attracted back again, and oedema resulted. The conception of Epstein was later confirmed by actual osmotic pressure measurements in a case of generalised oedema when it was found there was a pressure of

50 mm. of water in favour of filtration from blood to tissues—Krogh (1925). Leiter (1928) gave the first direct proof that oedema occurred in intact animals if the plasma proteins were experimentally reduced:—

> Healthy dogs were bled twice daily of 400 to 500 c.c. of blood. The blood was centrifuged and the corpuscles suspended in Ringer's solution re-injected. By this means the plasma proteins were gradually reduced without anaemia. On the fifth day oedema appeared accompanied by ascites, hydrothorax, and pulmonary oedema. By this time the plasma proteins had fallen from a normal of 7 gm. per cent. to 3 gm. per cent. and their osmotic pressure from 28 mm. to 15 mm. Hg.

I have seen the onset of oedema in cases of ascites after the peritoneal fluid had been removed by tapping. This was due to a rapid loss of proteins from the blood into the abdomen causing a fall in the level of the plasma proteins. In a large series of cases of nephritis and nephrosis with oedema, I have found the plasma proteins to be consistently reduced and their osmotic pressure to be below 15 mm. Hg.

In general terms, then, it may be said that whereas there are several other factors that influence the interchanges of fluid throughout the body, the plasma proteins play a very important part and stagnation of fluid in the tissues or nephrotic oedema appears when the colloid osmotic pressure of the plasma falls to 15 mm. Hg.

We now come to the second method of upsetting the fluid equilibrium of the body.

Raising the total osmotic pressure of the blood.—The following observations were made after the total osmotic pressure of blood had been raised by the intravenous injection of hypertonic solutions of 30 per cent. NaCl and 50 per cent. glucose—Robertson (1938). The amount given in each case was 5 c.c. per kg. at the rate of 2 c.c. per min. The following diagram shows the changes which occur in the haemoglobin after the injection of these solutions.

The line 00_1 in Fig. 3 represents the end of the injection and the space to the left of this line the time in minutes of the injection. The point on the line 00_1 joined by dotted lines represents the theoretical dilution of haemoglobin one would expect from an injection of 5 c.c. per kg. assuming the blood volume is 70 c.c. per kg. of body weight. You will note the theoretical dilution is 92 per cent. It will be seen that the dilution of haemoglobin was greatly in excess of 92 per cent., or the dilution caused simply by the addition of the volume of injected fluid. There must, therefore, have been a withdrawal of fluid from the fluid reserves throughout the body as a result of the hypertonic nature of the

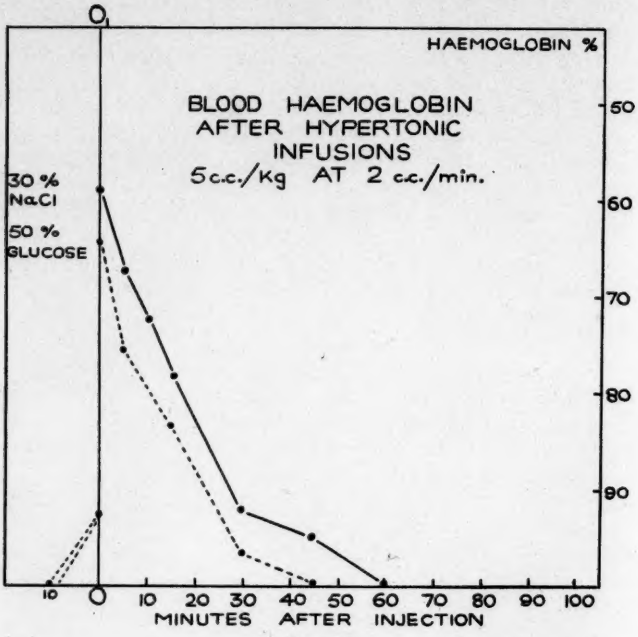

FIG. 3.

injection. Immediately the injection was completed the haemoglobin began at once to concentrate, indicating the vascular system was getting rid of the fluid that had been attracted to it. In about 30 to 45 minutes the haemoglobin was practically normal again. The main facts brought out by these experiments are:—

1. Hypertonic solutions such as 30 per cent. NaCl and 50 per cent. glucose attract a large volume of fluid from the tissue spaces producing a blood volume about twice normal.

2. The fluid is attracted from the tissues *only during the period of injection.*

3. The greatest blood volume is obtained the moment the injection is stopped, therefore the tissues are most dehydrated at that moment.

4. Immediately the injection is completed, the vascular system discharges fluid back to the tissues again and in $\frac{1}{2}$ to $\frac{3}{4}$ hour the blood volume is normal and the water content of the tissues is normal.

5. Lastly, these changes occur quite independently of the kidneys for similar findings are found after bilateral nephrectomy.

Briefly, these investigations on the fluid equilibrium in the body may be summarised:—

1. When the plasma proteins of the blood become depleted through any cause such as—

(*a*) Excessive loss.

(*b*) Destruction.

(*c*) Impaired production,

and their colloidal osmotic pressure falls from a normal of 25 mm. Hg to 15 mm. Hg or less, there is an increased production of tissue fluid or fluids produced by dialysis and this is made manifest by the presence of nephrotic oedema, hydrothorax, and ascites.

2. When the total osmotic pressure of the blood is suddenly raised by the intravenous injection of a hypertonic solution, *while the injection is taking place,* fluid is drawn into the vascular system very rapidly in an attempt to dilute the strong solution. So rapidly is this fluid transferred and equilibrium established that when the injection stops, the transfer of fluid from the tissues stops. At once fluid pours back from the vascular system to the tissues and in $\frac{1}{2}$ to $\frac{3}{4}$ hour the blood volume is normal and so is the fluid content of the tissues. In terms of tissue fluid therefore, we can say that after the injection of hypertonic solutions:—

(*a*) The tissue fluid volume is at a minimum the moment the injection stops.

(*b*) The tissue fluid volume is back to normal in $\frac{1}{2}$ to $\frac{3}{4}$ hour.

The behaviour of dialysates to changes in the fluid equilibrium is thus simply and easily defined.

What changes occur in the production of the aqueous humour when there is a disturbance in the formation of dialysates such as occurs in the pathological and experimental conditions that have just been described? First with regard to depletion of the plasma proteins.

Fall in the colloid osmotic pressure of the plasma.—It has been shown that the plasma proteins become very depleted in nephrosis and certain forms of nephritis, and that when this occurs generalised or nephrotic oedema results. I have made observations (Robertson, 1938) on the intra-ocular pressure in a series of thirty cases of nephritis or nephrosis with gross generalised oedema and I have found that the intra-ocular pressure measured with the Schiötz tonometer was within normal limits. It can be argued that the tonometer is not an instrument giving absolute and accurate measurements of the intra-ocular pressure, being influenced as it is by other conditions in the eye such as the elasticity, thickness, and circumference of the cornea. If the tonometric readings are taken by the same observer, however, on the same patient, with the same instrument, then the observations are of great relative value. A series of tonometric measurements

on individual cases throughout the period of oedema to recovery would therefore give valuable information. Lipoid nephrosis is a disease that may give such an opportunity and I have been able to carry out serial observations on a few cases of this rare disease.

Fig. 4 shows diagrammatically the findings in a typical case of lipoid nephrosis from the stage of oedema to that of complete

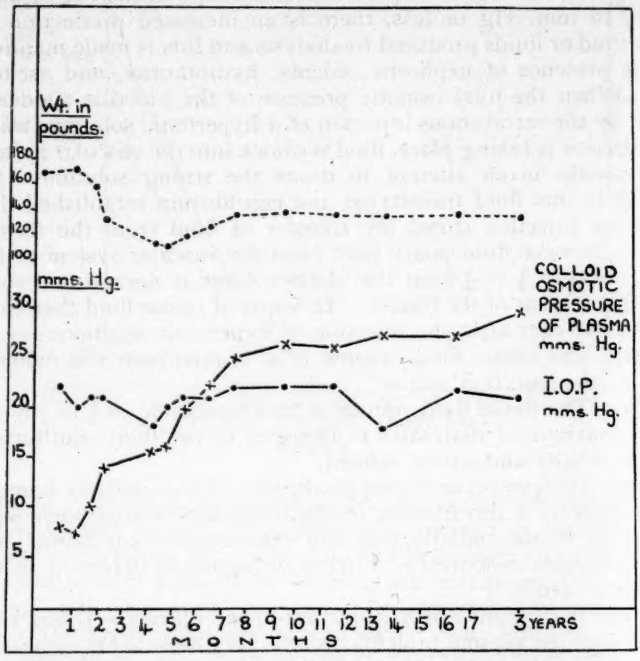

FIG. 4.

recovery. When first observed the patient had gross generalised oedema, ascites, and hydrothorax. Her urine contained 1 gm. of protein per cent. Under treatment the oedema gradually subsided and this chart demonstrates the changes noted during her illness.

1. *The Osmotic Pressure of the Plasma Proteins.*—When first observed the plasma proteins were so depleted that their osmotic pressure was only 7·5 mm. Hg as compared with a normal of 30 mm. Hg. On theoretical grounds, therefore, oedema was inevitable and it was in fact present in a marked degree. Gradually regeneration of the plasma proteins occurred and you will

see there was an increase in the colloidal osmotic pressure reaching a normal level about 9 to 12 months later.

Oedema.—The variations in the degree of water retention in the tissues is demonstrated very well by the weight chart. This patient's normal weight was 136 pounds. When first observed her weight was 169 pounds and as the oedema subsided her weight fell steadily to 112 pounds. There was thus 57 pounds of oedema fluid retained in the tissue spaces. At this point (112 pounds) she was free from oedema and we note that the osmotic pressure of the plasma proteins had risen above 15 mm. Hg. There is, therefore, a certain level of the colloid osmotic pressure of the plasma known as the *oedema-level* which lies just above 15 mm. Hg. Above this level—no oedema; below this level—oedema. The gain in weight after this point signified she was putting on flesh as clinical improvement took place and the plasma proteins continued to regenerate.

The Intra-ocular Pressure.—The intra-ocular pressure when first measured was 22 mm. Hg. It was within normal limits, and throughout the progress of this case no significant change was noted in the intra-ocular pressure which lay between 19 and 22 mm. Hg.

The conclusions we draw from these observations are that the eye does not share in the water-logging common to other tissues of the body. The equilibrium level of the intra-ocular pressure is not maintained by the hydrostatic pressure in the capillaries minus the difference in osmotic pressure between the aqueous humour and blood.

Let us now consider the eye when the second method of upsetting the fluid equilibrium is employed.

Raising suddenly the Total Osmotic Pressure of Blood.—Fig. 5 shows diagrammatically the changes which occurred in the intra-ocular pressure after the intravenous injection of 30 per cent. NaCl. The intra-ocular pressure was measured directly by the insertion of a cannula into the anterior chamber. During the blocked interval A the injection was given. After the injection was completed, there was a gradual fall in the intra-ocular pressure reaching its zero in about 60 minutes and remaining at this level for at least the next hour. The fall in the intra-ocular pressure was quite independent of the blood pressure. In terms of the volume of aqueous humour in the eye the curve of the intra-ocular pressure shows that as soon as the injection of NaCl was finished, the volume of aqueous began to diminish for the next hour, thereafter for the following hour at least the volume remained at a low level. How long it would remain subnormal I did not attempt to determine, but experience with 30 per cent. NaCl therapy in glaucoma shows the intra-ocular pressure may be kept

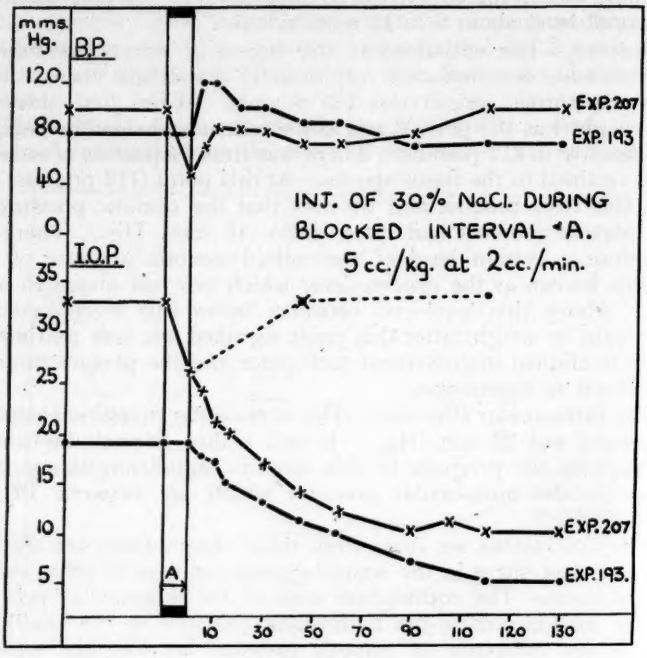

FIG. 5.

down for 24 to 48 hours. Put in another way, the injection of 30 per cent. NaCl stopped or greatly diminished the formation of the aqueous. How does the fluid exchange in the eye compare with that of the tissues? They differ markedly. If the intra-ocular fluid behaved like ordinary tissue fluid, then—

1. The greatest fall in the intra-ocular pressure should occur the moment the injection stopped, for you will remember the haemoglobin was at its dilutest and thus the transfer of fluid at its maximum.

2. At the end of the injection there should be a rapid climb back to normal in about 45 minutes. I have marked it " x " for you will remember at that time the haemoglobin was normal and the fluid content of the tissues was back to normal.

Fig. 6 shows diagrammatically the changes which occurred in the intra-ocular pressure after the intravenous injection of 50 per cent. glucose. During the blocked interval A the injection was given. It will be noted that no significant fall took place in the intra-ocular pressure. In other words the formation of aqueous

humour appeared to be little disturbed by the injection of 50 per cent. glucose. Once again the fluid exchange in the eye and the tissues differs; 50 per cent. glucose attracted a large volume of fluid from the tissues as evidenced by a great dilution of the haemoglobin, but none of that fluid came directly from the eye.

It is apparent that the eye does not behave as a simple fluid depôt as do the connective tissue spaces, and the aqueous humour

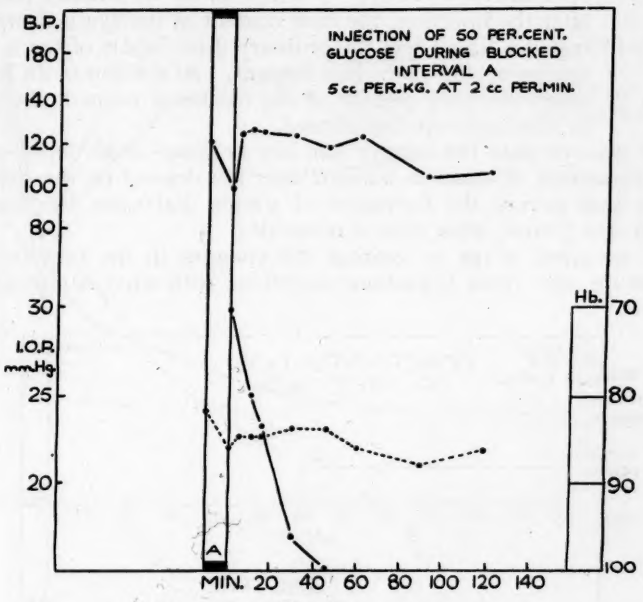

FIG. 6.

does not appear to be formed by the same process as the tissue fluid on the following grounds.

1. When the osmotic pressure of the plasma proteins in the blood falls below 15 mm. Hg and it can no longer counterbalance the hydrostatic pressure in the capillaries:—

(*a*) Nephrotic oedema results—*i.e.*, there is an excessive formation of tissue fluid.

(*b*) There is no increased production of aqueous humour.

2. Fifty per cent. glucose in doses of 5 c.c./kg., by reason of its hypertonicity withdraws a large volume of fluid from the tissues diluting the haemoglobin accordingly, but no fluid would appear to be withdrawn from the aqueous humour.

3. Thirty per cent. NaCl, which is $3\frac{1}{2}$ times stronger than 50

per cent. glucose dehydrates the tissue markedly. It also inhibits the formation of the aqueous humour; but the mechanism of dehydration of the tissues and that governing the inhibition of aqueous humour are not alike. Thus:—

(*a*) Dehydration of the tissues is greatest as the injection is being finished, and it has disappeared in ¾ hour.

(*b*) The inhibition of the formation of aqueous humour may continue for 24 hours. Further, in the 60 minutes period after the injection, the fluid content of the eye is decreasing at a time when the ordinary fluid depôts of the body are increasing their fluid content. At the end of an hour when the fluid content of the tissues is normal, the eye is most severely dehydrated.

If the eye does not behave like any ordinary fluid depôt, and the formation of aqueous humour does not depend on the simple laws that govern the formation of simple dialysates like tissue fluid and lymph, what does it resemble?

It occurred to me to compare the changes in the intra-ocular pressure after these hypertonic injections with what happened to

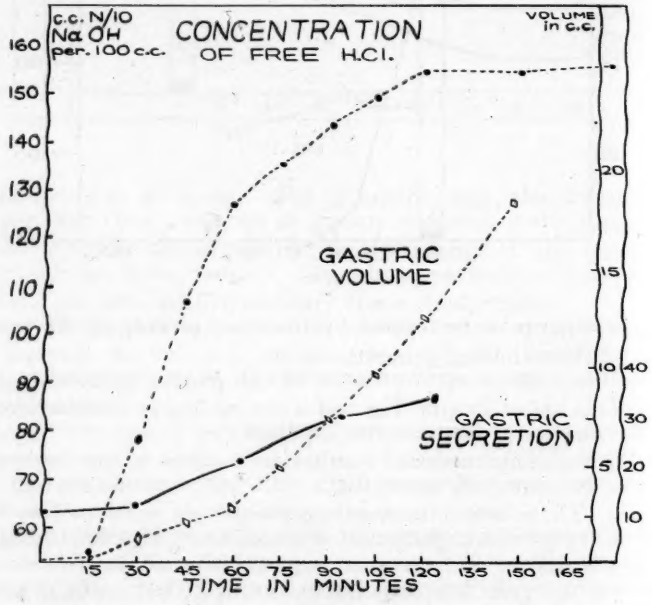

FIG. 7.

Gastric secretion after histamine stimulation.

a generally accepted secretion. The gastric secretion was chosen as one easily studied.

Diagram 7 shows the curves of the secretion of free HCl and the volume of gastric juice produced in cats after the subcutaneous injections of histamine 1 mg. hourly. In addition, there is plotted the gastric secretion expressed as a factor—the product of total acid and volume over 100. This curve is a mean of six normal

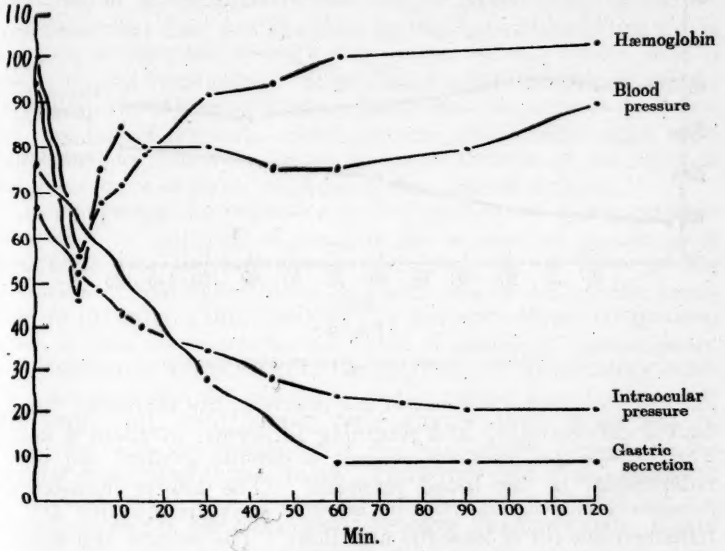

FIG. 8.*

Effect of 30 per cent. NaCl intravenously.

controls. In the subsequent figures the gastric secretion factor alone will be plotted to avoid confusion with other observations such as blood-pressure, intra-ocular pressure, and haemoglobin which were taken simultaneously. We note that a rapid rise in the concentration of free HCl occurred reaching a maximum of 160 c.c. N/10 NaOH and being maintained at that level indefinitely. The volume of gastric juice is being secreted at a uniformly constant rate of 6 to 10 c.c. an hour. The gastric secretion factor shows a gradual rise.

Effect of 30 per cent. NaCl—5 c.c./kg. at 2 c.c./min.—It will be seen from Fig. 9 that the haemoglobin as before became

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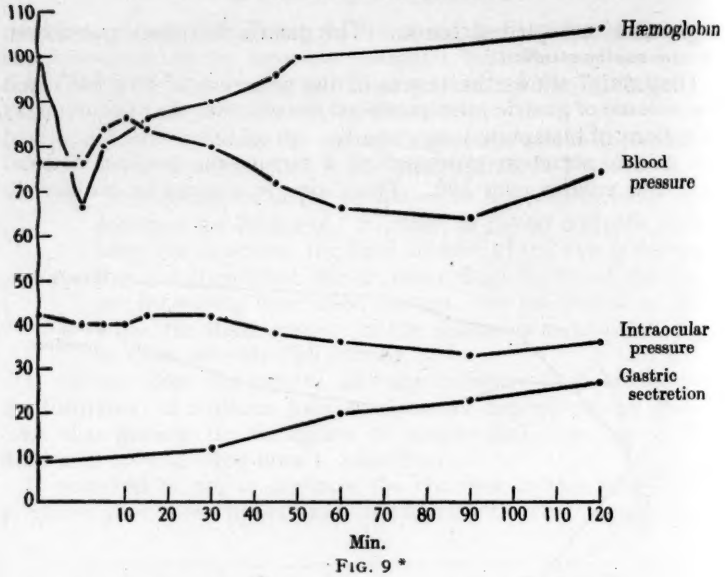

FIG. 9 *

Effect of 50 per cent. glucose intravenously.

markedly diluted at the end of the injection, but thereafter immediately concentrating and returning to normal in about ¾ hour. The intra-ocular pressure showed a definite gradual fall quite independent of the blood pressure. The lowest intra-ocular pressure was reached about 90 minutes after the injection and it remained low for at least the next hour. The volume and acidity of the gastric juice were both lowered (in some experiments complete inhibition for 3 to 4 hours occurred) and this is demonstrated by a gradual fall in the gastric secretion factor. If we consider that the stomach is receiving a continuous stimulation with histamine, the most powerful gastric stimulant known, then it is apparent that the inhibiting action of hypertonic NaCl must be great.

Effect of 50 per cent. glucose—5 c.c./kg. at 2 c.c./min.—As in previous experiments it will be seen from Fig. 9 that the haemoglobin became diluted at the end of the injection, then rapidly returned to normal. However, there was usually no effect on either the intra-ocular pressure or on the gastric secretion.

In comparing the gastric secretion with the intra-ocular pressure it is important to decide exactly how much information the intra-ocular pressure gives about the formation of the aqueous humour.

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Assuming the blood pressure is constant, the intra-ocular pressure is a mean of the volume of aqueous produced and that eliminated. Therefore, a fall in the intra-ocular pressure may be due to:—

1. Diminished production.
2. Increased elimination.
3. A combination of these two.

During the injection of 30 per cent. NaCl it is probable that any fall which occurs in the intra-ocular pressure is due to an increased elimination of the aqueous humour, for Weed (1922) found the cerebro-spinal fluid was absorbed by two routes in addition to the normal mechanism through the arachnoid villi under similar experimental conditions. Immediately after completion of the injection, osmotic equilibrium is established throughout the body, so any fall in the intra-ocular pressure which takes place after this must be due to a failure in the production of the aqueous by some more complex mechanism than one of dialysis.

In comparing the stomach and the eye, it would appear perhaps less open to criticism to compare the volume of gastric juice produced with the intra-ocular pressure, and the curve of the secretion of free hydrochloric acid with that of the specific secretion in the eye. Fortunately for the purposes of our comparison now, it does not matter for the curve of volume of gastric secretion intimately follows that of the curve of free hydrochloric acid. We see then that the behaviour of the gastric secretion after 30 per cent. NaCl and 50 per cent. glucose bears some sort of relationship to the behaviour of the intra-ocular pressure under similar experimental conditions. Thirty per cent. NaCl inhibited gastric secretion and during that period of inhibition of the gastric secretion, the intra-ocular pressure gradually fell. Again 50 per cent. glucose had no effect at all on the gastric secretion nor had it any on the intra-ocular pressure.

From the above experiments it would appear therefore that the formation of the aqueous humour in many respects does not resemble that of the so-called interstitial fluid like lymph, pleural or peritoneal fluids. In many respects the control of the equilibrium level of the intra-ocular pressure which is related to the formation of the aqueous humour, resembles the control of the gastric secretion.

So far a study has been made of the means by which the equilibrium level of the intra-ocular pressure can or cannot be altered after artificial variations have been made in the osmotic pressure of the blood, and there would appear to be evidence that the behaviour of the eye cannot be explained adequately by the simple laws which govern dialysis.

In conclusion I would like to discuss certain aspects of the eye which have a bearing on its fluid equilibrium and these are—

1. The question whether the aqueous humour circulates.
2. Where the aqueous humour is formed.
3. Where the aqueous humour is absorbed.

The Circulation of the Aqueous Humour.—In the same way as there has been controversy regarding the mechanism of the formation of the aqueous humour, so it has been stated by various observers that there was no circulation whatsoever, and that there was a circulation. The views of these different schools of thought are important for their respective beliefs influenced them in their theories on how the aqueous humour was formed. The supporters of the non-circulation or stagnant aqueous theory maintained that the only movement present was an interchange of water and ions across the capillary membrane all around the eye. The nature of this simple interchange of fluid has already been described in Fig. 1 showing a normally functioning capillary. All the movement that occurs is a to and fro one. The supporters of the circulation theory believed that there was a definite through and through circulation from the posterior chamber to the angle of the anterior chamber. The supporters of the stagnant aqueous maintained it was formed by a process of dialysis or ultra-filtration, whereas the supporters of the circulating aqueous maintained it was formed by a process of secretion or transudation.

The evidence now would appear to be more strongly in favour of the view that the aqueous humour does circulate, but curiously enough the acceptance of this view by certain workers did not prevent them at the same time still supporting the theory that the aqueous was produced by dialysis and thus theoretically stagnant. The evidence that a circulation exists is supported by observations of Priestley Smith (1927) some time ago, and more recently by Friedenwald and Pierce (1931). To give one or two examples:—

1. There is the condition of iris bombé, where an iridectomy restores a channel from the posterior to the anterior chamber with relief of the secondary glaucoma.

2. Cells which were cast off from a melanosarcoma of the iris have been shown by microscopic studies to be lodged in the tissues around the angle of the anterior chamber throughout its circumference.

3. A case has been described of a child with very small lenses. In one eye on one occasion the lens slipped over the pupil and formed with the iris a complete barrier between the anterior and posterior chamber. The iris slowly bulged forward around the edge of the lens and the anterior chamber became evacuated. As soon as the iris approached the cornea the intra-ocular tension began to rise.

It has been calculated by Friedenwald and Pierce that the rate

of flow of the aqueous humour is in the region of 1 c.mm. per minute.

The site of formation of the aqueous humour.—To be consistent the upholders of the dialysate theory must, as I believe they do, maintain that the aqueous humour is produced with equal freedom by diffusion through the capillaries of all the intra-ocular vessels of the ciliary processes, iris, and retina. Because of the through and through circulation, however, it would seem that the formation of fluid must take place in the posterior chamber; and of the structures in the posterior chamber evidence points to the ciliary processes as being the probable site of the production of the fluid. There is first of all their histological structure; the lack of fluid which results from their removal or cutting off of their blood supply, and the absence of changes in the intra-ocular pressure after removal or absence of the iris or arrest in the blood supply of the retina.

The site of removal of the aqueous humour.—To be consistent upholders of the dialysate theory should maintain that the aqueous humour is absorbed through the same capillaries as it is produced for one of the characteristics of the capillary membrane is its reciprocal permeability. There is evidence, however, that no absorption of aqueous humour can take place from the posterior chamber—*i.e.*, the type of membrane only allows a flow of fluid in one direction. The aqueous humour can only escape through the walls of the anterior chamber, and there seems ample evidence to show that the greatest, if not the only exit, is through the canal of Schlemm.

How does the fluid get into the canal of Schlemm? That is a question which is not easy to answer. Very little indeed is known with certainty about the nature, structure, function and contents of this most important canal.

Contents.—It is now generally accepted that under normal conditions it contains aqueous humour, but in venous stasis it may contain blood due to backflow from veins.

Connections.—It has no direct communication with the anterior chamber and is separated from it by a layer of endothelium. It communicates freely with the anterior ciliary veins into which the aqueous eventually empties.

Structure.—It has not the structure of a vein, but closely resembles a lymphatic channel. It is certainly not a vascular capillary as has been several times referred. A vessel which (*a*) allows fluid traffic in one direction only; (*b*) does not normally contain red cells; and (*c*) begins as a cul-de-sac so to speak and enters the venous system, cannot be a vascular capillary.

The fluid, therefore, cannot enter the canal by osmosis, for not being a capillary there is no plasma there to absorb it. It is

not known with certainty whether the pressure in the canal of Schlemm is greater, equal to, or less than that of the intra-ocular pressure. It may be that the canal pressure is not static and that at times it may fall below the intra-ocular pressure to allow the aqueous to enter it by simple filtration; thence by propulsion to be sent on to the veins. Various methods have been described to explain the mechanism of the exit of the aqueous humour, but none has given complete satisfaction.

The conclusions from a further study of the fluid equilibrium of the body and of the eye are similar to the views expressed by me previously—Robertson (1937) (1938).

1. The formation of the aqueous humour is not governed by the same simple laws that govern the lymph, pleural and peritoneal fluids and other dialysates. Dialysis is therefore not a satisfactory explanation of the production of the aqueous humour.

2. When the osmotic equilibrium in the body is disturbed in various ways the fluid formed in the stomach and the eye are disturbed rather similarly. This suggests that a secretory process in the eye may play some part in controlling the intra-ocular pressure.

3. Ample evidence is available that the aqueous humour circulates from the posterior to the anterior chamber.

4. Evidence seems to point to the site of formation of the aqueous humour being in the ciliary process.

5. The aqueous humour leaves the eye at the angle of the anterior chamber into Schlemm's canal by some process which is not osmosis, and no fluid can leave the eye normally by the posterior chamber.

REFERENCES

BENNETT, DODDS and ROBERTSON (1931).—*Quart. Jl. Med.*, Vol. XXIV, p. 239.

DRINKER and FIELD (1933).—Lymphatics, Lymph and Tissue Fluid. Baillière, Tindall & Cox., London.

EPSTEIN (1914).—*Jl. exp. Med.*, Vol, XX, p. 334.

——— (1917).—*Jl. Amer. Med, Assoc.*, Vol. LXIX, p. 444.

——— (1917*a*).—*Amer. Jl. Med. Sci.*, Vol. CLIV, p. 638.

——— (1922).—*Amer. Jl. Med. Sci.*, Vol. CLXIII, p. 167.

FRIEDENWALD and PIERCE (1931).—*Bull. Johns Hopkins Hosp.*, Vol. XLIX, p. 259.

——— (1932).—*Arch. of Ophthal.*, Vol. VII, p. 538.

KROGH (1925).—Anatomy and Physiology of the Capillaries (New Haven), p. 265.

LANDERER (1884).—Die Gewebsspannung in ihrem Einfluss auf die Ortliche Blut— und Lymphbewegung. Vogel, Leipzig.

LANDIS (1930).—*Amer. Jl. Physiol.*, Vol. XCIII, p. 353.

LEITER (1928).—*Proc. Soc. Exp. Biol. and Med.*, Vol. XXVI, p. 172.

SMITH, PRIESTLEY (1927).—*Brit. Jl. of Ophthal.*, Vol. XI, p. 263.

ROBERTSON (1937).—*Brit. Jl. of Ophthal.*, Vol. XXI, p. 401.

——— (1938).—*Brit. Jl. exp. Path.*, Vol. XIX, p. 30.

——— (1938).—*Lancet*, Vol. I, p. 1435.

——— (1938).—*Brit. Jl. of Ophthal.*, Vol. XXII, p. 79.

WEED (1922).—*Amer. Jl. Anat.*, Vol. XXXI, p. 191.

ANNOTATION

One-eyed Drivers

A report on one-eyed drivers appeared in the issue of the *Sight-saving Review* of November, 1938, by three Research Associates in the Institute of Human Relations at Yale University. It appears that in the United States one to two per cent. of drivers are totally blind in one eye, while between 20 and 40 per cent. have a deficient eye which handicaps them. No State prohibits either group from driving. "Only about 20 States make any check on vision for the driver's licence, and there is no agreement regarding visual standards." The authors of this report conclude that "most one-eyed and deficient-eyed drivers are oblivious to the defects which predispose them to accidents. For most of these drivers the solution lies in personal re-education adapted to their individual needs, for the greatest human hazard arises not from the defects but from ignorance of the dangers from such defects."

The one-eyed driver is particularly handicapped in night driving. He is more sensitive to glare and it takes him longer to recover from the glare and to see the road clearly again. The loss of binocular field can be compensated to a certain degree by keeping the head turned at a small angle, but the position is to a certain extent awkward and strained and would be likely, we think, to lead to fatigue of the neck muscles. "Another form of correction possible is to keep the head and the eyes roving continually from right to left while driving in order to bring possible dangers from the side into the field of vision of the good eye."

The report ends with tables of hazards to which the left-eyed and right-eyed persons are exposed. It must be remembered that in the United States the driver sits on the left-hand side and drives on the right side of the road.

We have no hesitation in commenting on vision in motoring in spite of an annotation on this subject last year. The matter is of the greatest importance in view of the tragedy of the roads. That there is nothing to stop the one-eyed driver from obtaining a licence seems to us to indicate that the question of vision was not sufficiently considered when the rules were drawn up. The fact that Hannibal, who was uniocular, crossed the Alps, cannot be considered an argument in favour of the one-eyed driver, for we believe his form of transport was the elephant, which if as powerful in some respects as the modern motor engine certainly never achieved anything like the speed attained at the present day. We are inclined to think that a one-eyed person should not be granted a licence. With regard to the driver with one defective eye we think that the advantage

of a binocular field probably compensates for lack of macular acuity and we see no reason why such a person should be debarred from driving. This report will have done good if it calls the attention of the authorities in the States to the importance of the subject, and we will end with the pious hope that it will soon be as difficult to find a one-eyed driver in America as it was to find a dead postboy in the old coaching days in England.

ABSTRACTS

I.—GLAUCOMA

(1) **Magitot, A.—Glaucoma without rise of pressure. (Glaucomes sans hypertension).** *Ann. d'Ocul.*, Vol. CLXXV, p. 349, May, 1938.

(1) **Magitot** is of opinion, that the disease which A. v. Graefe has labelled amaurosis with excavation, should be classified into three groups:—

(*a*) Myopic glaucoma according to the conceptions of Axenfeld.

(*b*) Very chronic glaucoma with only occasional rise of the intra-ocular pressure. The disc may or may not show cupping.

(*c*) A third group, which presents a deep atrophic cupping. Here, the word glaucoma is probably a misnomer, and the old name amaurosis with excavation is more adequate. Pressure of the sclerosed carotids on the chiasma or on the optic nerves has been suspected as the cause of this atrophy. However, the author is not fully satisfied with Thiel's radiographic evidence of sclerosis of the intracranial carotids. The proof is still wanting, that sclerosis of the carotids or of the anterior cerebral artery does result in atrophic cupping. Many old people, whose carotids offer the same radiographic aspect, do not show an atrophic excavation. Further, tumours of the hypophysis undoubtedly exercise a considerable pressure on the chiasma and the optic nerves; nevertheless, the consequent optic atrophy is only exceptionally associated with excavation.

HUMPHREY NEAME.

(2) **Malling, B. (Bergen).—Investigations on the so-called glaucoma capsulare. (Einige Untersuchungen ueber das sogenannte Kapselglaukom).** *Acta Ophthal.*, Vol. XVI, p. 43, 1938.

(2) **Malling** investigated 181 patients suffering from glaucoma simplex and found that the deposits on the posterior aspect of the cornea which are regarded as characteristic of capsular glaucoma,

were seen frequently in simple glaucoma, though not as frequently as in capsular glaucoma. Occasionally they were also found in apparently normal eyes. He subjected a series of eyes in which corneal deposits were present with or without changes in the lens capsule and with or without glaucoma, to the compression test (*i.e.*, rate of recovery of the tension of the eye reduced by compression with a tonometer or such like instrument). He found that in most of the cases the channels of excretion must be patent, and therefore dismisses exfoliation of the lens capsule and blocking of the angle with its debris as the primary cause, and argues that the corneal deposits, the lens exfoliation and the glaucoma are all signs of an uveal lesion. Each of these three signs may be seen singly. The changes in the capsule must be regarded not as the cause of glaucoma, but as a sign of an uveal affection that may lead to it.

ARNOLD SORSBY.

(3) **Dalsgaard-Nielsen, E.—Glaucoma-like cupping of the optic disc and its aetiology.** *Acta Ophthal.*, Vol. XV, p. 151, 1937.

(3) **Dalsgaard-Neilsen** draws attention to those cases of cupping of the disc which have all the appearances of glaucoma except that increase of tension cannot be demonstrated. She recalls the work of Ransom Pickard and other observers in assessing the significance of these appearances, and reports 12 personal cases. In five of these, detailed studies of tension, fields and provocative tests revealed chronic glaucoma. In three more the diagnosis of glaucoma was complicated by the associated traumatic or arteriosclerotic lesions. In two the cupping was unquestionably due to pressure atrophy from a pituitary tumour. In one case the appearances were probably of congenital origin and in the final case no aetiology could be established. The water tolerance test proved helpful in this study and radiography of the internal carotids was useful in establishing arteriosclerosis as a possible cause—as, of course, also the presence of a pituitary tumour.

ARNOLD SORSBY.

(4) **Hausmann, G. (Vienna).—The late results of cyclodialysis. (Zur Kenntniss der Dauerresultate der Zyklodialyse).** *Zeitschr. f. Augenheilk.*, Vol. XCII, p. 139, 1937.

(4) From a statistical analysis of 105 cases out of 326 treated by cyclodialysis at Meller's clinic in 1920-1935, **Hausmann** gives the following results:—

(1) Increase of tension after cataract extraction:

(*a*) After uncomplicated extraction: good results in eight out of 11 cases.

(*b*) After complicated extraction: good result in one out of two cases.

(2) *Secondary glaucoma: seven cases, five successfully treated.*

(3) Primary glaucoma treated by cyclodialysis only: in 34 cases *(47 eyes), 28 cases (30 eyes) showed satisfactory results.*

(4) Primary glaucoma treated by cyclodialysis and subsequently *another operation: 22 cases (31 eyes).* The second operation (further cyclodialysis or iridectomy, etc.) normalised tension *in 13 cases (18 eyes).*

(5) Primary glaucoma treated by cyclodialysis subsequent to *a previous glaucoma operation. Twenty-one cases:* of these 15 had iridectomy in the first place and four (6 eyes) *trephine operation. Except in two iridectomy cases cyclo-*dialysis normalised the tension. The value of cyclodialysis *as a second operation is stressed.*

ARNOLD SORSBY.

II.—MISCELLANEOUS

(1) **Jameson Evans, J. (Birmingham).—The syndrome of the oculo-nasal nerve neurosis.** *Birmingham Med. Rev.*, March, 1938.

(1) **Jameson Evans'** paper was read at a meeting of the Birmingham Branch of the British Medical Association on November 17, 1937. After a brief outline of the anatomy of the nasociliary nerve he gives notes on five cases which he considers more or less typical of oculo-nasal neurosis. In analysing these cases he finds that the main ocular lesions are three in number. First, sectorial epithelial dystrophy of the cornea and conjunctiva, marginal heaping and oedema of the corneal epithelium with fine translucent elevations or crinkling which at times erode to form punctate superficial ulcers; these heal with very little scarring, occasionally a more central type of infiltration may leave a definite nebula. Second, sectorial or complete anaesthesia or hypoaesthesia of the cornea. Third, episcleral and conjunctival vasodilatation, usually in sector form, corresponding to the corneal involvement.

The nasal lesions are apparently neither pathognomonic nor specific. But it can be stated that gross focal sepsis in the nose does not as a rule give rise to the oculo-nasal neurosis. The nasal lesions found are not as a rule acutely inflammatory or purulent. Nasal discharge is watery, the turbinates are congested and oedematous. Deflected septa are not unusual. The nasal lesions are thus in the anterior part of the nares.

The author's hypothesis as to the way these lesions in the eye arise is as follows:—noxious efferent impulses are set up by the nasal lesion and travel up the oculo-nasal nerve till they reach the

long ciliary nerve. Here they are diverted down the long ciliary nerve, antidromically, to the eye.

Treatment is directed to the nasal condition by means of local anaesthetics combined with adrenalin, and 0·5 per cent. phenol. Surgical measures are only rarely indicated but zinc ionisation can be given a trial if thought desirable.

The ocular lesions need simple soothing applications, such as shade or pad; phenolaine, yellow oxide of mercury ointment. In the case of stubborn ulcers of the cornea atropine is indicated, but it is most unusual to have to resort to cauterisation of the ulcer in this connexion.

R. R. J.

(2) **Miklós, Andor (Debrecen).—Experiences with the tension-relieving therapy in tabetic optic atrophy. (Erfahrungen mit der "Entlastungstherapie" bei tabetischer Sehnervatrophie).** *Arch. f. Ophthal.*, Vol. CXXXVIII, p. 219.

(2) The method of treatment of tabetic optic atrophy employed by **Miklós** was based on the theory, already put forward by other writers, that a fall in the general blood pressure is frequently seen in tabetics; this affects the retinal circulation with consequent disturbance in the nutrition of the retina and a rapid decline in the vision and field.

Treatment was accordingly directed to an improvement in the circulation of the retina (*a*) directly, by raising the general blood pressure by the administration of tonics, such as strychnine and caffein (although their effect is comparatively slight), alcohol and tobacco in moderation being allowed, and (*b*) indirectly, by reducing the tension with miotics and operation.

Operation—cyclodialysis as modified by Blaskovics—was the most effective method. Pilocarpine did not uniformly reduce the tension, but was most useful in prolonging the effect of the operation or as a prophylactic in early cases of abnormally low blood pressure.

The results, here recorded, of this treatment, which was combined with the energetic use of anti-syphilitic drugs, were so good that it is recommended as worthy of trial.

THOS. SNOWBALL.

(3) **Linksz, A. (Budapest).—Dermotherapy in the everyday practice of oculists. (Dermatotherapie in der alltäglichen Praxis des Augenarztes).** *Klin. Monatsbl. f. Augenheilk.*, Vol. XCVIII, p. 433, 1937.

(3) In chronic inflammatory conditions of the eyelids a number of remedies are often tried in turn without success. **Linksz** gives a careful survey of the treatment of lid conditions with special reference

to the qualities of the skin which may be met with in different individuals. He gives details of prescriptions with the dermatological indications for their proper use. For these the original paper should be consulted.

D. R. CAMPBELL.

(4) **Aliquo-Mazzei (Arezzo).—The ocular signs of invasion of the base of the skull by pharyngeal tumours. (*Manifestazioni* oculari nei tumori della base del cranio di origine faringea).** *Bull. d'Ocul.*, March, 1938.

(4) The differential diagnosis of these tumours, which are not common, is difficult. The author gives the story of three cases which have come under his observation and discusses the symptoms and the means of diagnosis.

The nature of these tumours is various; about half are carcinomatous and half sarcomatous. In the latter the mucous membrane of the pharynx retains its normal appearance; in the former, it is changed. The first symptoms are usually slight; often deafness is the most marked, since the tumour may begin near the Eustachian tube. Later, as it invades the base of the skull it may cause severe headache, or trigeminal neuralgia, if it invades the fifth nerve at its exits. Sooner or later there is usually some interference with the movements of the eye; the sixth nerve and the third being attacked in that order. Careful examination by radiography in several positions of the head will give indications of the mode of invasion of the skull. At least three projections are necessary; axial, lateral and submento-vertical. The posterior clinoid processes are always eroded. Barré considers this finding as pathognomonic of these tumours. Treatment by surgical interference is useless in **Mazzei's** opinion. Treatment by radium and X-rays seldom gives permanent benefit but often relieves the pain for a time.

HAROLD GRIMSDALE.

(5) **Bietti (Rome).—The action of drugs on the pressure in the central retinal artery. (Ricerche sull'azione di alcuni farmaci sulla pressione dell'arteria centrale della retina con particolare riguardo all' adrenalina).** *Riv. Oto.-Neuro.-Oftal.*, March-April, 1938.

(5) It has often been assumed that the action of drugs, such as adrenalin, is the same for every part of the body. This is certainly not true in every case. **Bietti** has made a long series of experiments to establish the action of these drugs in man. He shows that their action differs in different regions of the body. Small doses of adrenalin given subcutaneously are followed by a marked fall in the pressure in the arteria centralis especially in the diastolic pressure;

very small doses given intravenously (1/80 to 1/120 mgr.) sometimes cause a similar fall, but less constantly. Larger doses always are followed by a rise of pressure; sympatol has an action parallel to that of adrenalin. The action of caffeine, which is known to dilate the vessels of the brain, may provoke a fall in the retinal pressure, but less than that after adrenalin. Certain bodies which are generally antagonistic in action to adrenalin, also, may cause a fall in the arterial pressure in the retina; such for example follows the use of large doses of lymphoganglin and less constantly of doses of ergotamine.

The author thinks that the fall in pressure following subcutaneous injection of adrenalin is principally due to passive dilatation of the vessels following vasoconstriction of the general vessels. He thinks that the behaviour of the retinal arteries may be an index of the reaction of the cerebral arteries.

HAROLD GRIMSDALE.

(6) **Dandy, Walter E. (Baltimore).—Intra-cranial aneurysm of the internal carotid artery cured by operation.**

Matas, Rudolph (New Orleans).—Aneurysms of the circle of Willis. *Annals of Surgery*, Vol. CVII, No. 5, May, 1938.

(6) **Dandy's** case is, as far as he is aware, the first attempt to cure an aneurysm at the circle of Willis by direct attack upon the aneurysm.

A frail, small, sallow man, of 43 years of age, came to the Johns Hopkins Dispensary in mid February, 1937. He was afflicted with complete paralysis of the right oculomotor nerve. He had been in hospital for three months in 1936 for gastric disturbance, possibly ulcer, and he had been drinking heavily for the past 18 months.

The present attack began six days before admission with severe frontal pain. Intense stabbing pain was complained of in the right eye the same day and he slept badly that night. Next morning he was aware of diplopia and later in the day the right eye closed.

On admission the only positive finding was complete paralysis of the right third cranial nerve. Fundi, fields and reflexes were all normal. An X-ray examination of the head revealed no abnormality. The patient attended the Dispensary for five weeks without improvement and was then referred to Dandy. The Wassermann reaction was negative.

Dandy operated on March 23, 1937, using a small hypophyseal approach. Marked cortical atrophy was present, evidenced by the pools of fluid in the subarachnoid spaces, the removal of which fluid gave ample room for exposure. An aneurysm, the size of a pea, was found on the outer wall of the internal carotid, adjacent to the entry of the posterior communicating artery. The aneurysm did not

involve this artery but arose from the carotid by a narrow neck. The third nerve passed obliquely backwards in its normal course and was attached to the aneurysm at one point only, viz., where it entered the cavernous sinus. There was no evidence of subarachnoid bleeding.

Forceps placed upon the thick aneurysmal wall pulsated forcibly. An ordinary flat silver clip was placed over the neck of the sac and tightly compressed, obliterating it completely. The clip was flush with the wall of the carotid artery. The sac, lateral to the clip, was then picked up with forceps and thrombosed by the electrocautery. It shrivelled to a thin shred of tissue.

The post-operative course was attended by an attack of delirium tremens, but the man made a good recovery and left hospital in two weeks time. Seven months later there was complete return of all functions referable to the third nerve.

Dandy, in discussing this case, refers to a similar case which he saw ten years earlier in which the internal carotid was ligatured in the neck, but the patient died of cerebral softening.

This interesting case is illustrated by photographs of the patient before and 13 days and 7 months after the operation; two X-ray pictures of the skull and an excellent diagram of the aneurysm and the parts concerned.

Matas's paper is a long contribution which he gave as a discussion on Dandy's case, with supplementary remarks. It is too long to abstract here and is accompanied by a bibliography of 62 items. Both papers should be read in the original.

R. R. J.

(7) **Orzalesi and Cassuto (Florence).—The action of sympatol on the pressure in the arteria centralis retinae. (L'azione del sympatol sulla pressione nell'arteria centrale della retina).** *Boll. d'Ocul.*, April, 1938.

(7) It has been shown that the cerebral vessels, and the ocular which are derived from the cerebral, react to certain vaso-active drugs in a different way from those in other regions of the body. Thus, for example, amyl nitrite which produces a lowering of pressure generally, provokes an increase in the arteria centralis retinae. Sympatol, the substance which is the subject of this paper, has a chemical structure very similar to that of adrenalin, and is very similar in action.

In all cases, both when given by instillation and subcutaneously, a considerable reduction of the arterial pressure in the retina was noted. In all, this was greatest after about twenty minutes and the pressure returned to its former height in about two hours. In no case was there a rise. In this it seems to agree with adrenalin

(which endovenously sometimes showed an increase, according to Bietti, but in the present series of experiments intravenous injection does not seem to have been tried).

HAROLD GRIMSDALE.

(8) **Baratta (Parma).—The action of sympamin (Phenyl-isopropyl-amine) on the human eye. (Ricerche sperimentali sull'azione della simpamina (fenil-isopropil-amina) sull'occhio umano).** *Boll. d'Ocul.*, May, 1938.

(8) Several papers have been reviewed in this Journal recently on the action on the eye of substances allied to adrenalin. **Baratta** has investigated the action of sympamin in man. This substance, sold commercially as "benzedrin," has been largely used especially in America, on account of its astringent action on mucous membranes. The author finds that instillation of sympamin hydrochlorate produces mydriasis which begins 10 minutes after application and is at maximum in about 20 minutes. The mydriasis lessens after 90 minutes and passes off in about 6 to 8 hours. The effect of subconjunctival injection is similar. He has found that the addition of sympamin to cocaine increased the anaesthetic action and lengthened it. If the sympamin was dropped in at an interval after the cocaine, there was no reinforcement. Similar reinforcement was seen when the drug was added to other mydriatics, homatropine and atropine. Alone, it seems to have no effect on the intra-ocular pressure; its value therefore, to assist ophthalmoscopic examination is probably considerable.

HAROLD GRIMSDALE.

(9) **Cordero (Pisa).—The action of *a*-dinitrophenol on the eye. (L'azione dell' *a*-dinitrofenolo sull'occhio).** *Arch. di Ottal.*, pp. 152, 213 and 294, 1937.

(9) The action of certain drugs, used to decrease obesity, has been studied recently on account of their other more dangerous actions, since in some cases they have proved fatal. These drugs which act by increasing metabolism and raising the body temperature belong to the aromatic series. The first accurate knowledge on their thermogenetic power is due to Cazeneve and Lapine who used this property to raise the temperature of animals on which they were making experiments. Later, in the years of the War, it was found that men who worked in the preparation of phenol nitrate were often poisoned and sometimes died. It was noted that there was great rise of temperature with rapid breathing. The amount of oxygen consumed was largely increased. It seems that the drug does not cause this hyperthermia by means of the centres of the medulla since destruction of this region does not prevent the rise of temperature. The action, therefore, seems to be peripheral rather than central, but the exact course cannot be yet ascertained.

It is an important practical point that thyroid seems to sensitise towards the nitro-derivatives; those patients who begin a course of dinitrophenol after taking thyroid, are more easily poisoned by the latter. When the patient complains of weakness, headache, or profuse sweating, erythematous eruptions, or yellow colouration of the skin, these are danger signals and the drug must be stopped immediately. In some of the fatal cases agranulocytic anaemia has followed these premonitory symptoms.

In so far as the eye is concerned, the only damage noted is the development of cataract; this has followed in a considerable proportion of cases. The subjects are most commonly women, probably because men are less anxious to diminish their girth. The cataract comes on usually after some months of treatment with the drug, but sometimes at a time after treatment has been suspended; in one recorded case the interval was nine months. The beginning is usually the development of numerous vacuoles, close under the anterior capsule; then follow opacities in the same position, which are often coloured. In the later stages the nucleus appears gold-yellow or copper coloured. This appearance is diagnostic of this form of cataract; the history of treatment by *a*-dinitrophenol is usually obtainable and helpful. In some cases the lens swells much during the formation of the cataract and may cause increase of tension. In many cases operation has been performed without complication.

HAROLD GRIMSDALE.

(10) **Cordero (Pisa).—The action of *a*-dinitrophenol on the eye. (L'azione dell' *a*-dinitrofenolo sull'occhio).** *Arch. di Ottal.*, May-June, 1938.

(10) In this number **Cordero** continues his study of the action of this drug; with the idea of finding some connection between the lesions of general health and the special lesions which affect the eye, he has examined the various organs of the animals, the subjects of his experiments both macro- and microscopically. He finds that when there is acute poisoning the symptoms resemble those of strychnine. All the animals showed signs of acute emphysema, with small haemorrhages and oedema of the lungs; early inflammatory changes in the kidneys; and slight fatty degeneration of the heart muscle. These animals died within a few hours of the first injection.

Others were kept alive, with smaller doses, for a considerable time; some for more than a year; immediately after every injection a rise of temperature and of the rate of breathing was noted; this soon passed off. Generally there was a fall in weight but one rabbit gained considerably; this animal lived for 13 months under treatment. The rise of temperature in this experiment was small,

never exceeding a degree and a half; in many cases the daily average temperature was raised about half a degree.

Abscesses were noted in various parts of the body: the contents were caseous; the skin over them ulcerated; these healed easily, if the drug were stopped; neither in the cases of acute poisoning nor in the chronic, were any changes observable in the eyes. As in the acute cases, the organs most damaged are the lungs, the kidneys, the liver and the heart.

HAROLD GRIMSDALE.

(11) **Magitot, A. and Dubois-Poulsen (Paris).—Condemnation of coloured test objects in clinical perimetry. (Condemnation des index colorés en périmétrie clinique).** *Ann. d'Ocul.*, Vol. CLXXIV, p. 649, October, 1937.

(11) **Magitot** and **Dubois-Poulsen** give a survey of the difficulties which may be encountered when coloured test objects are used. These difficulties may be due either to the method of examination or to the condition of the patient.

Difficulties of the first type include the changing contrast phenomena, the hue, brightness and variability of the test objects. A small red object will appear grey in the periphery of the visual field. On its way to the centre a yellow colour will be perceived before red can be seen. Accordingly, the contrast phenomena will change and influence the visibility of the object. Therefore, it has been recommended to paint the perimeter in a grey of the same brightness as that presented by the test object. Thus there will be no contrast phenomenon, the visibility will be improved and the visual field will appear to be larger. Further, it is difficult to obtain test colours which do not become darker in a rather short time, especially if the colours are not very saturated. There is the following dilemma. To get exact perimetric fields spectral lights would be required. On the other hand a full field can be traced only in a bright illumination, which will tend to desaturate the colours.

In addition to these difficulties, the colour deficiences of the patient have to be taken into account. Of course, coloured test objects are of little use in taking the field of a patient who has an abnormal colour sense. Further, the colour absorption by the lens in old age, the better focusing through a narrow pupil, the restriction of the field for red in hyperopia, for blue in myopia has to be considered.

Intelligence and attention come much more into play when coloured test objects are used. It requires a certain training to appreciate the intermediate tints in the periphery. Fatigue comes on much more quickly than in working with white test objects and makes it useless to examine a patient for longer than ten minutes.

The principal use of coloured test objects is to disclose in a rapid way the existence of a central scotoma.

There is no relative colour scotoma. An absolute scotoma will be found in such cases by sufficiently small white objects. According to the authors the Bjerrum screen is not superior to the 33 cm. perimeter for exploring the central field, if care is taken to damp down contrasts, to keep the illumination constant and to use sufficiently small objects. The central area is best examined by the stereoscopic method or by Evans' angioscotometer, the zone between ten and thirty degrees by a 1 mm. object and the peripheral field by a 3 mm. object. The test object should not be mounted on a handle as the patient might become aware of any movement of the surgeon. The projection of a spot light on the perimetric arc constitutes an excellent test object.

The superiority of "white" perimetry is illustrated by some cases in which red test objects failed to reveal peripheral islands or important characteristic features of the field. Repeated examinations by white objects gave an average variation of 24 per cent. against 108 per cent. when red objects were used.

HUMPHREY NEAME.

(12) **Streiff, E. B. and Zeltner, C. (Paris).—The syndrome of Laurence-Moon-Bardet-Biedl (pigmentation of the retina, polydactyly with Fröhlich's syndrome and mental deficiency. (Le Syndrome de Laurence-Moon-Bardet-Biedl (Rétinite pigmentaire, polydactylie, distrophie adipose-génitale, deficience mentale). (Etude clinique et génetique).** *Arch. d'Ophtal.*, Tome 2, p. 289, 1938.

(12) A long paper with many genealogical tables and having particular reference to the associated eye symptoms:—Errors of refraction, strabismus, retinitis, hemianopsia, together with alterations in the vitreous and the crystalline lens.

The authors point out the probability of the associated clinical symptoms being due to changes in the diencephalic hypophysis.

An extensive bibliography is appended.

FRANCIS R. HILL.

(13) **Coppez, H. and Dujardin, B. (Brussels).—A case of Besnier-Boeck's disease or Schaumann's benign lymphogranulomatosis with ocular manifestations. (Un cas de maladie de Besnier-Boeck ou lymphogranulomatose bénigne de Schaumann a manifestations oculaires).** *Arch. d'Ophtal.*, Tome 2, p. 497, 1938.

(13) After describing the general symptoms of the disease and commenting upon the doubt which still surrounds its aetiology, the authors refer to the resemblance that it bears in some aspects to syphilis; while in the stage of generalisation the local lesions react to the tuberculin tests.

They then describe a case under their own care and review the ocular symptoms noted by other observers in similar cases, particularly iridocyclitis.

FRANCIS R. HILL.

(14) **Magitot, A. (Paris).—Ocular pain and its treatment by anaesthesia of the spheno-palatine ganglion and orbital injection of alcohol. (La douleur oculaire. Sa thérapeutique par l'anesthésie du ganglion sphéno-palatin et l'alcoolisation orbitaire).** *Ann. d'Ocul.*, Vol. CLXXIV, p. 361, June, 1937.

(14) **Magitot** continues a series of papers which discuss in great detail the importance of the anaesthesia of the spheno-palatine and ciliary ganglia in ocular affections, with special reference to the treatment of inflammatory conditions of the anterior segment and uncompensated glaucoma. The efficiency of this treatment is due to damping down of the hyperactivity of sympathetic fibres. Thus, profuse lacrimation (the lacrymal gland is supplied by facial fibres which originate in the sympathetic intermediate portion of Wrisberg and run to the spheno-palatine ganglion), blepharospasm and photophobia can be eliminated.

The anaesthesia of the spheno-palatine ganglion by painting the posterior part of the middle turbinate with Bonain's lotion containing equal parts of cocaine, phenol and menthol is not so reliable as the anaesthesia of this ganglion by an injection of novocaine into the pterygo-palatine canal from the mouth. Novocaine has only a very transient effect. For an anaesthesia of longer duration the injection of 40-60 per cent. alcohol is recommended. The needle should have rather a short point in order to avoid a lesion of the palatine vessels, an accident which might be followed by necrosis of the maxillary bone.

A far superior method is the anaesthesia of the ciliary ganglion by retrobulbar injection of 1 c.c. 1 per cent. novocaine followed after half-a-minute by 1·5 c.c. alcohol. Again, the needle should be short-pointed to avoid the orbital veins. The only untoward after-effect seen was a transient paresis of the external rectus. To prevent this inconvenience, the injection should be made neither too far outwards nor inwards.

These injections do not only relieve pain. They have a far reaching therapeutic effect which is most striking in interstitial keratitis. The photophobia disappears and the cornea becomes clear within a month in cases which can be expected to take more than four times longer.

The orbital injection transforms an acute or subacute glaucoma into a chronic one, allowing surgical interference to be done under more favourable conditions. However, the operation should not be

postponed for long as the tension remains high though the pain has ceased. It is interesting to see the corneal oedema disappear while the hypertension persists after the orbital injection. The author claims therefore that the usual view regarding the corneal oedema as a sequel to the increased tension is wrong. Further, orbital injections of alcohol have an established value in the treatment of iritis and iridocyclitis, especially in hypertensive and very acute cases. It is striking to see a patient suffering from gonococcal iritis relieved by the injection.

The corneal sensation returns 48 hours after the injection, but the pain does not. The injections may be repeated weekly.

HUMPHREY NEAME.

CORRESPONDENCE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—In his second letter, *Brit. Jl. of Ophthal.*, December, 1938, Professor v. Liebermann refers to his paper: "Kaustische Resektion der trachomatoesen Bindehaut," published in the *Arch. f. Ophthal.*, Vol. CV, pp. 542-550, 1921. From this paper it may be understood that this operation was applied by him in 22 cases of cicatricial trachoma, and in 14 other cases this operation was combined with tarsectomy—but till now not a single case of operation of a severe case of spring catarrh by this method has been published by him.

In 1922, in the *Klin. Monatsbl. f. Augenheilk.*, Vol. LXVIII, p. 617, Prof. v. Liebermann wrote that he proposed to perform this operation in suitable severe cases of spring catarrh, and even gave to this proposed operation the name: "Kaustische Resektion der Tarsalbindehaut bei Conjunctivitis Vernalis"—but so far, evidently this proposition was not carried out, since no operation in a severe case of spring catarrh by his method was published by him.

Basing myself on 32 cases of severe spring catarrh operated on by my method, I may say that the caustic resection proposed by Professor Liebermann is not applicable in cases of severe spring catarrh. From the Fig. on page 546, *Arch. f. Ophthal.*, which demonstrates his operation, I understand that the three sutures drawing the everted tarsus, are lying on the surface of the conjunctiva, 1·5 mm. from the border of the lid. These sutures must prevent the destruction by galvanocautery of the papillae of spring catarrh,

"which sometimes have grown so violently that they turn up to the intermarginal line forming such a thick layer of conjunctiva that it is 2-3 times thicker than the tarsus lying under it." (See my paper, p. 288.) Deep galvanocautery in such cases must destroy the whole border of the lid together with the lashes, and make an unmendable deformity of the lid itself.

I take the liberty of asking Prof. Liebermann to read my operation in full in the original, and not only the short abstract in the *Klin. Monatsbl. f. Augenheilk.*, January, 1938, Vol. C, p. 112, of the paper read by me in the XV Concilium Ophthalmol. On the basis of this short abstract he claims his priority also in the same volume of *Klin. Monatsbl. f. Augenheilk.*, p. 469. If he will do so I hope it will be clear that my operation is entirely different from his "proposed caustic resection," which I repeat was never performed by him in severe cases of spring catarrh.

In conclusion I permit myself to publish below Professor A. Trantas', of Athens, letter to me of 28/8/38 concerning the priority of Professor v. Liebermann in the above-named operation; it is essential to mention that Professor v. Liebermann, in the *Klin. Monatsbl. f. Augenheilk.*, 1922, strongly criticises the operation of Trantas, which is similar to my idea, but about which I never heard before.

"Cette opération et la vôtre se ressemblent, mais il est incontestable que nous avons travaillé tous les deux indépendemment l'un de l'autre. C'est pour cela que je vous félicite pour votre initiative. Quant à Mr. Liebermann, il a tort, car, comme vous le dites bien, son procédé est tout autre et encore parce que lui-même reconnait qu'il ne l'avait pas essayé dans aucun cas du catarrh printanier, et encore parce que ma communication a été faite le 1919, tandisque lui-même le 1921, il n'avait pas pratiqué encore aucune opération sur le catarrh printanier, ni même la destruction de la conjonctive tarsale par le galvanocautère. Dans le travail du 1920 du Liebermann il s'agissait de l'application de sa cautérisation et recouvrement par le cul de sac dans le trachome et non dans le catarrh printanier.

Je répète que j'ai senti un réel plaisir parce que vous aussi avez eu la même idée indépendemment de moi."

Sincerely yours,

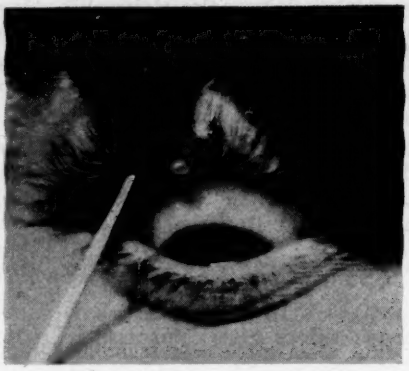
Dr. N. I. SHIMKIN.

HAIFA, *December* 14, 1938.

RHINOSPORIDIOSIS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS,—In August of last year you were good enough to publish a paper of mine entitled "Rhinosporidiosis of the Eye." In view of the fact that I have had numerous letters from various ophthalmic surgeons interested in the subject I would like you to print a photograph of my case who now shows a recurrence at the muco-



cutaneous junction of the right upper lid. The area of bulbar conjunctiva from which the previous polypus was removed twelve months ago shows no activity and the cornea has remained clear. The photograph being enlarged just shows the yellowish-white specks in the substance of the polypus.

I should be very grateful if any of your readers know of any local treatment besides excision, which will guard against recurrence of the polypi in different parts of the conjunctival sac in which, recurrences only seem to show after an incubation period of at least twelve months.

I am, yours truly,

HERBERT KAYE.

JOHANNESBURG, *December* 1, 1938.

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS,—It will be of interest to some of the readers of the *Brit. Jl. of Ophthal.* to learn that the short review of the important monograph on the pathology of trachoma by Professor Birch-Hirschfeld, published in the issue of December, 1938, p. 762, will

be supplemented by an extended abstract in a future number of the *International Review of Trachoma.*

As a result of this monograph Pulvertaft and MacCallan now have withdrawn the view they expressed as to the origin of the bleb-like excrescences in the second stage of trachoma (Tr. 11a) published in the *Lancet* of January 25, 1936, p. 215, and elsewhere.

Yours faithfully,

A. F. MACCALLAN.

RESEARCH LABORATORIES,
WESTMINSTER HOSPITAL MEDICAL SCHOOL,
LONDON. *January 9, 1939.*

NOTES

Oxford Ophthalmological Congress

THE dates for the next meeting of the Oxford Ophthalmological Congress are July 6 to 8 inclusive. The new Master of the Congress is Mr. Percival J. Hay.

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Honour

AMONG the New Year's honours we are pleased to see the name of Lieut.-Colonel E. O'G. Kirwan, our Calcutta representative, who receives the C.I.E.

* * * *

Appointments

Mr. P. MCG. MOFFATT and Mr. A. S. PHILPS have been elected Assistant Surgeons to the Royal Westminster Ophthalmic Hospital.

Mr. J. MINTON has been elected Ophthalmic Surgeon to out-patients at the Hampstead General Hospital.

* * * *

Royal London Ophthalmic Hospital

THE Annual Dinner of Past and Present Students of the Royal London Ophthalmic Hospital, will be held at the Langham Hotel, Portland Place, W.1, on Thursday, March 9th, 1939, at 7 for 7.30 p.m. Chairman:—A. F. MacCallan, Esq., C.B.E., M.D., F.R.C.S., Consulting Ophthalmic Surgeon to Westminster Hospital.

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The de Schweinitz Lecture

A LECTURE in honour of Dr. George E. de Schweinitz has been founded by the Section on Ophthalmology of the College of Physicians of Philadelphia, to be known as the de Schweinitz Lecture. The lecture is to be given annually on the third Thursday of November, on a subject to be chosen by the speaker selected.

The Ziegler Prize

THROUGH the generosity of Mrs. Kathryn Ziegler Halsey and S. Lewis Ziegler, Jr., the S. Lewis Ziegler prize has been established by the Section on Ophthalmology of the College of Physicians of Philadelphia. The sum of one hundred dollars is annually offered for an original contribution to ophthalmology deemed of sufficient merit for an award of the prize; the Committee of Award to be appointed by the Chairman of the Section on Ophthalmology. Theses for the 1938-39 award must be in the hands of the Committee by October 1st, 1939.

For particulars, communicate with Dr. Warren S. Reese, 1901 Walnut Street, Philadelphia, Pa.

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Lectures at Glasgow

OWING to unforeseen circumstances the lecture by Professor Emile de Grósz has been postponed until Tuesday, April 25, 1939.

FUTURE ARRANGEMENTS

1939

February 3.—North of England Ophthalmological Society, at Newcastle-upon-Tyne.

February 3.—Midland Ophthalmological Society, at Oxford Eye Hospital.

February 10.—Section of Ophthalmology, Royal Society of Medicine.

March 3.—North of England Ophthalmological Society, at Liverpool.

March 10.—Section of Ophthalmology, Royal Society of Medicine. (Clinical Meeting.)

March 31.—North of England Ophthalmological Society, at Sheffield. (Joint Meeting with the Midland Ophthalmological Society.)

April 20-22.—Ophthalmological Society of the United Kingdom, Annual Congress, at the Royal Society of Medicine.

May 4-5.—Irish Ophthalmological Society, Dublin.

May 19.—Midland Ophthalmological Society, at Gloucester Royal Infirmary and Eye Institution.

June 9.—Section of Ophthalmology, Royal Society of Medicine. (Annual.)

July 6-8.—Oxford Ophthalmological Congress, at Oxford.

October 6.—Midland Ophthalmological Society, at Birmingham and Midland Eye Hospital. (Annual.)

CONTEMPORARY OPHTHALMIC LITERATURE

Archives of Ophthalmology. *December, 1938.*

LANCASTER. Aniseikonia.
BRÜCKNER. Physiologic and clinical ophthalmologic problems in relation to individual variability.
GUY. Simple dacryocystorhinostomy.
MCDONALD and LIPPINCOTT. Angiomatosis retinae; report of a case with a pathologic study of the enucleated eye.
LEMOINE. Lesion of the optic tract probably the result of infected sphenoid sinuses.
SWINDLE. Events of vascularization and devascularization seen in corneae.
KNAPP. Orbital hyperostosis: its occurrence in two cases of meningioma of the skull.
JONES, JORDAN and SULLIVAN. Intra-ocular nematode worms: report of a case and review of the literature.
VERHOEFF and KING. Scleromalacia perforans: report of a case in which the eye was examined microscopically.
BELLOWS, PUNTENNEY and COWEN. Use of sorbitol in glaucoma.

Annales d'Oculistique. *November, 1938.*

MAGITOT. Recent and partial idiopathic retinal detachment.
WEVE and FISCHER. The clinical value of proteolytic ferments in sub-retinal liquid.
WEVE and FISCHER. The importance of pH for the proteolytic activity of sub-retinal liquids and its inhibition.
WEVE and FISCHER. The metabolism of the detached retina.
WEVE and FISCHER. The degeneration of the albuminoid matter in retinal detachment.
SÉDAN. Recurrent limbal epithelioma with large palpebral and corneal insertions distinctly separated.

Revue Internationale du Trachome. *October, 1938.*

COSMETATOS and CHARAMIS. The fight against trachoma in Greece.
CORNET. The symptoms of trachoma described by Keller in 1937.

Archiv. für Ophthalmologie. *December, 1938.*

GREEFF. What picture have we of Albrecht von Graefe? Part IV.
SIEGERT. Melanosarcoma of the iris with particular consideration of the growth and process of development.
KINUKAWA. Two cases of eclamptic retinitis (gravidarum) with pathological and anatomical findings.
SALIT. The mineral stabilitity of cataractous and sclerosed human lenses.
ZOBEL. Corneal sensation in various periods of life.
BAKKER. The significance of the iris in the development of infra-red cataract.
DIMITRIOU. The relation of tabetic optic atrophy to the alterations of the general blood pressure and the intra-ocular pressure.
PODESTRA, HERMANN and BAUCKE. Where does vitamin C come in the different tissues of the eye?
KOYANAGI. Proliferating tumour of the retinal pigment epithelium associated with a metastatic choroidal carcinoma.
KLEMENS. The occurrence of cystoid degeneration in the retinal periphery and the suppression of the newly formed vascular layer.
ZIERING. Squint eye function and squint deviation.
LEYDHECKER. A family with congenital microphthalmos.
TATAR. Glaucoma in Cushing's disease.
HOFE. Fundus changes in diabetics.

OHM. What is dark? Observations on Dr. van Essen's essay, "Conception and significance of the visual sensation of dark."
VAN ESSEN. The essence of dark. Also an answer to the observations of Professor Ohm on my theory of visual dark.

Klinische Monatsblätter für Augenheilkunde. *November, 1938.*

URBANEK. Vitamin C metabolism in cataract.
REISER. Experiences with intra-capsular cataract extraction.
VOGT. First histological condition of lens capsule praecipitate.
VOGT. Further clinical and histological findings of senile anterior capsule desquamation.
MEYER. Essential conjunctival shrinkage or pemphigus conjunctivae.
BRUHN. Clinical and experimental researches on ocular injury.
JAEGER. Operative treatment of pterygia.

Zeitschrift für Augenheilkunde. *October, 1938.*

KENTGENS. Vitamin A therapy in corneal disease.
RINTELEN. Further results of the blood pressure determination in retinal vessels.
RADNOT. Carcinoma of the Meibomian glands.
ASCHER. Epithelial budding in the anterior chamber. Regressive changes and healing after Rontgen rays.
KRAUPA. Dot and ring form of corneal degeneration.

November, 1938.

AMSLER. Javal's frustrated keratoconus.
STREIFF. Adaptation of Nordenson's apparatus to keratoscopy and photography of the anterior segment of the eye.
GOLDMANN. The technique of slit-lamp microscopy.
BANGERTER. An unusual case of diabetic cataract.

Bollettino d'Oculistica. *October, 1938.*

STREIFF. The beaded pigmentary stria of chorio-retinitis (Siegrist).
BELLO. Experimental research on the natural histogenous immunity of the conjunctiva in rabbits against pneumococci, gonococci and diphtheritic bacilli.
SALA. A case of paralysis of the upward movement of the eye with rare associated symptoms.
CIOTOLA. Microphthalmos and malformation of the digits (syndactyly, polydactyly).
DI BARI. Tolerance in the constancy of spectacles.
ANGELLO. Angioneurotic oedema and allergic conjunctivitis.

Archivos de Oftalmologia de Buenos Aires. *September, 1938.*

ORTIZ. Experimental action of galactose on the lens of a dog.
TISCORNIA, JUST AND MERCANDINO. Rare evolution of an ethmoidal mucocele and its spread into the lacrymal passage.
ALLENLE. Observations on varicose conjunctival vessels, corneal ulceration, corneal leucoma and astigmatism.

October, 1938.

HURTAULT and SVERDLICK. Extraction "in toto" of a complicated cataract with the organized pupillary membrane.
ADROGUE and LAGOS. Strabismus. A case of permanent post-operative diplopia.
TETTAMANTI. Parenchymatous degeneration of the transparent cornea.
SATANOWSKY and CRAMER. Another case of dictyoma of the ciliary body.
PEYRET. Mooren's rodent ulcer.
SVERDLICK. Presentation of a lamp for post images and of an apparatus of similar type to Tschermak's. Study of sensorial correspondence and functional strabismus.